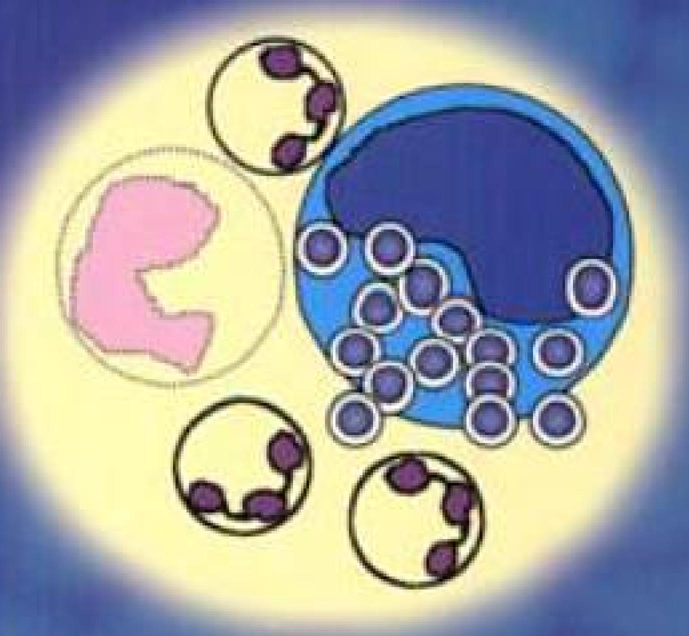

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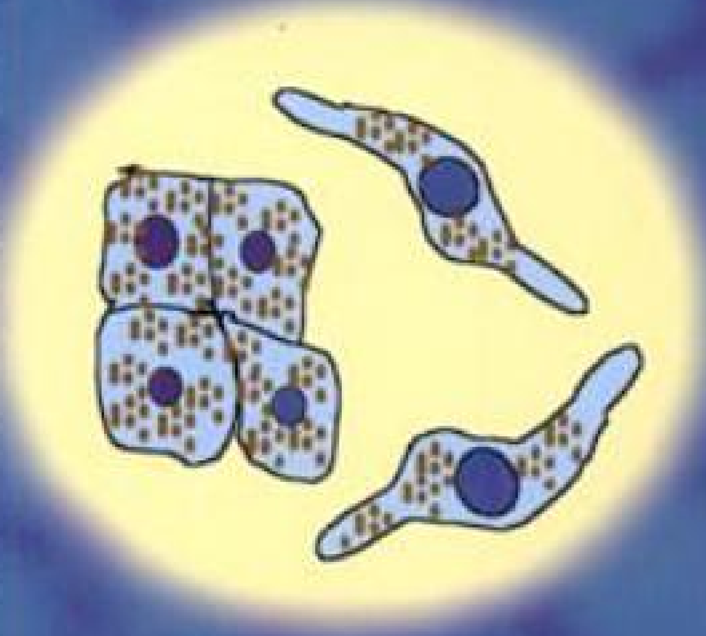
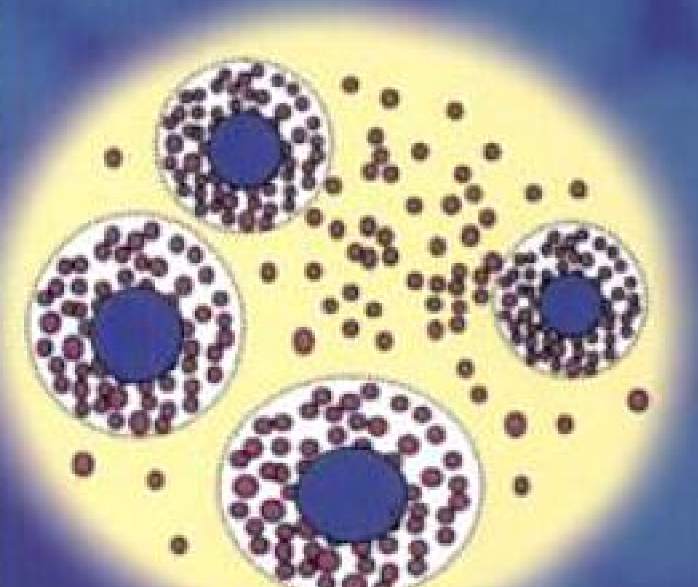


Small Animal Dermatology

a practical guide to the diagnosis and management of skin diseases in dogs and cats



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Small Animal Dermatology

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Small Animal Dermatology

A practical guide to the diagnosis and management of skin diseases in dogs and cats

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Preface

Skin disease is one of the most common reasons for dogs and cats to be presented to veterinarians and yet dermatological diagnosis and treatment can often prove troublesome. In theory, the approach to the diagnosis and treatment of skin conditions should be no different to that used for other body systems. Clinicians must first make a diagnosis (or tentative diagnosis) and then prescribe treatment. However, dermatological diagnosis is complicated by the fact that the skin has only a limited range of responses to a wide variety of cutaneous insults. Hence, the appearance of different diseases may be similar. Furthermore, multiple conditions may be present in the same animal causing overlap of clinical signs. The evaluation of treatment is also difficult because various agents may have a therapeutic effect without achieving clinical cure. What appears to be preliminary success can soon become failure.

To avoid these problems, clinicians must use a logical and systematic approach when approaching skin diseases. The steps in this approach are:

- Taking a history
- Performing a physical examination
- Identifying the predominant problems
- Making a diagnosis or generating a list of differential diagnoses
- Performing tests to rule in or out the differential diagnoses (if a diagnosis has not already been made)
- Prescribing treatment for the disease, or diseases, diagnosed
- Re-inspecting the animal to monitor the response to treatment

This book aims to provide the reader with an easy-to-use guide on working through this process. Chapter 1 provides guidelines on how to take appropriate histories and make the most of the information obtained. In Chapter 2, details are given on how to perform a dermatological examination and recognise the significance of any skin lesions that occur. Based on the information obtained from the history and physical examination, the practitioner can then use Chapter 3 to decide what is the main presenting problem, and which of the following chapters to consult:

- Chapter 4 – Pruritus
- Chapter 5 – Alopecia
- Chapter 6 – Scaling, crusting and seborrhoea
- Chapter 7 – Macular, papular and pustular diseases
- Chapter 8 – Draining tracts and non-healing wounds

- Chapter 9 – Erosive and ulcerative diseases
- Chapter 10 – Pigmentary changes
- Chapter 11 – Lumps and swellings
- Chapter 12 – Otitis

The use of this problem-oriented approach greatly simplifies the diagnostic process because the skin diseases are categorised according to how they present to the clinician. Each of these chapters begins with a list of differential diagnoses for the problem. Then, for each condition listed, a lesion distribution diagram is shown along with key clinical findings. The relevant tests that can be used to make a specific diagnosis are also indicated. At the end of each chapter, a general diagnostic approach to each problem is presented in the form of an algorithm so that the practitioner can use the relevant tests in a logical sequence throughout the investigation of the case.

Chapter 13 provides specific instructions on how to perform and interpret the various diagnostic tests that are used in veterinary dermatology. For each test, a step-by-step 'recipe-style' guide is provided that highlights the techniques needed to ensure good results. The conditions that can be diagnosed with each test are listed, along with an indication as to the degree of difficulty associated with the technique and its interpretation. Numerous examples of the potential results that may occur with each test provide a comprehensive guide to its diagnostic interpretation.

After making a specific diagnosis (or diagnoses), the clinician can find details of specific therapy in Chapter 14. In this chapter, the objectives and basic principles of dermatological treatment are described, followed by a 'formulary-style' guide to the products that can be used. Each section ends by describing why the treatment may not be working, and what to do about it.

It is hoped that this book will prove useful to veterinary students who are just beginning to learn about skin diseases, but also for practitioners who wish to improve their diagnostic accuracy when dealing with these very common presentations. The aim is to have produced a book that is practical enough to sit in the consulting room where it can be readily consulted on a daily basis.

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This book is dedicated to my family and Sarah, the new member. It has only been possible because my teachers in veterinary dermatology shared their knowledge. Thank you Gus Walton, Geoff Shawcross, Danny Scott, Doug DeBoer, Karen Moriello, Wayne Rosencrantz, Craig Griffin, Keith Thoday and Richard Halliwell.

1

History taking

Taking an accurate and detailed history is the first step in any diagnostic investigation and this is especially important for skin problems. Pet owners can provide more historical information about skin conditions than for any other body system. This is because the clinical signs of skin disease are easily visible and the owner can actually see lesions develop and change over time. The information obtained from the owner is vital for formulating differential diagnoses, an essential step prior to the performance of tests or initiation of treatment.

How to ask questions

Before starting to take histories, clinicians must learn 'how' to ask questions. Mastering this aspect of history taking is an important skill that requires practice and experience. Without it, the clinician will not be able to obtain the relevant information from the client. The way in which questions are asked is important because it constitutes one aspect of the veterinarian's 'bedside manner'. This is critical for building the client's confidence and ensuring subsequent compliance throughout the diagnostic and treatment phases of the condition. During the first few minutes of a consultation, the veterinarian must strike up an immediate professional relationship with the client. In this critical phase the clinician must appear to be friendly, caring, compassionate, conversational and competent, all at the same time! Veterinary students should observe a number of experienced practitioners to learn the skills required to be successful in this multi-faceted role.

In addition to the qualities mentioned above, clinicians must be very logical and objective when questioning clients. They need to extract information from the client that might not otherwise be forthcoming. Clients may not mention important facts because they are not aware of their relevance. They may also be embarrassed at disclosing information about previous home remedies or conditions involving neglect. If the clinician is not entirely convinced by a particular response, it is often helpful to repeat the question in a slightly different way to determine if the answers are consistent. Useful information can also be obtained by listening to other people in the examination room such as the client's partner or children.

In order to take a history to investigate a skin problem, the veterinarian must become familiar with dermatological vocabulary. However, it is important that clinicians do not speak to clients using technical terminology that is not easily understood. The clinician must become 'bilingual', using plain language for clients, and veterinary terminology for professional colleagues and medical records. To help students of veterinary medicine with this important process, Table 1.1 provides a list of terms that are used to describe skin conditions, along with a suitable 'translation' that most clients would likely use or understand. Precise definitions of these terms are provided later in the text when they are encountered.

Table 1.1 Common dermatological terms used when taking histories

Dermatological term	Laypersons' term
Pruritus	Itch
Alopecia	Hair loss or baldness
Lesion	Area of abnormal skin
Scaling	Dandruff
Seborrhoea	Dandruff or greasy skin
Crust	Scab
Comedone	Blackhead
Erythematous macule	Spot or blemish
Papule	Spot or pimple
Pustule	Spot
Macular-papular-pustular eruption	Rash
Hyperpigmentation	Skin has turned black
Hyperpigmented macule	Freckle
Hypopigmentation	Skin or hair has turned pale or white
Lichenification	Skin has become thicker
Vesicle	Blister
Erosion	Looks like an abrasion
Ulcer	Red, raw skin
Fissure	Split or crack
Nodule	Lump
Otitis	Inflamed or infected ear

What type of history to take

If any meaningful information is to be obtained from the history, it is important for the clinician to know what questions to ask, and the relevance of the answers given by the client. This is a difficult aspect of history taking to learn because it requires a sound knowledge of dermatological conditions as well as general medicine.

There are three basic types of history that can be used to evaluate skin diseases:

1. The partial history
2. The complete history
3. The recheck history.

The partial history

The partial history involves asking a few focused questions about the specific skin problem presented by the owner and only generates limited information. Although this type of history taking is commonly employed by veterinary surgeons, it should be avoided except for cases that fulfil the following criteria:

- The diagnosis is immediately obvious as soon as the client mentions the problem and the animal is seen. Examples of this would be the cat that has been fighting and has developed an abscess on its face, or the dog that has suddenly started scratching and is covered in fleas.
- The animal is already well known by the veterinary surgeon and its general health status is monitored regularly.

The complete history

The complete history provides comprehensive details of the animal's dermatological problem as well as its general health status (see below). Ideally, this type of history should be obtained at the onset of any skin condition. In practice, this can be difficult to achieve due to time constraints. Nevertheless, the clinician should be aware of how to perform a complete history and should utilise this approach in any case in which a diagnosis can not easily be made at the first consultation. For the busy practitioner, it may be necessary to arrange an extended or double appointment for more complex cases so that this essential information can be gathered. Alternatively, but not as satisfactory, the complete history can be put together over a series of appointments.

The recheck history

It is not necessary to obtain a complete history when re-evaluating animals that are undergoing treatment. This is because most of the information will be the same as that obtained during the initial consultation. Hence, when performing a recheck history, the clinician should focus on two main aspects:

- Is the treatment working?
- Have there been any problems or adverse effects associated with the treatment?

Taking a complete history

The components of a complete dermatological and general medical history are summarised in Table 1.2. This information should be committed to memory so that questions are not forgotten during the consultation. Some clinicians prefer to start with the dermatological history whereas others prefer to obtain the general medical history first. This is purely a matter of preference and is not important as long as the clinician decides on a particular system and uses it routinely. To help with this process, some veterinary dermatologists convert the information in the table into a questionnaire that can be filled in by the client prior to entering the examination room. Other clinicians use the table as a checklist that is completed during the consultation.

The relevance of the information obtained during the complete history is as follows:

Patient details

The basic patient details will normally be known for animals that are already registered with a practice. If not, this information should be obtained before the start of the main consultation. Some of this information, especially the signalment (age, sex, breed), is very important in helping to generate differential diagnoses.

Age of the animal

It is important to know the animal's age because some skin disorders can show age-related onset. Congenital and hereditary diseases usually occur in young animals whereas metabolic and neoplastic diseases are more likely in older animals. Table 1.3 lists some diseases that typically show age-related onset. Note that this may not be the age at which the animal presents for the problem.

Table 1.2 The components of a complete dermatological and medical history

Patient Details	
Signalment	Age Sex Breed
Origin of the animal	Age when acquired Place where acquired
Vaccination status	Primary course and boosters
Worming status	Frequency and type of treatment
Presenting problem	
Owner's main complaint	Owner to describe
Dermatological history	
Nature and duration of the condition	How long has the problem been present What was the condition like at the beginning How has the problem changed over time
Further specific information about pruritus	Distribution Severity Onset relative to other lesions
Contagion	Other animals People
Familial history	Sire, dam or siblings
Previous treatments and response	Treatment from other veterinary surgeons Home remedies
Diet	What does the animal eat
Environment	Where does the animal live Have there been any changes
General medical history	
General activity level and exercise tolerance	Normal or decreased (lethargic)
Body weight	Increased or decreased
Appetite	Increased (polyphagia) or decreased (inappetance or anorexia)
Thirst	Increased (polydipsia) or decreased
Cardio-respiratory system	Coughing or sneezing
Gastrointestinal system	Vomiting or diarrhoea Abnormal appearance of stools
Urinary system	Changes in frequency Abnormal appearance of urine
Reproductive system	Normal libido or oestrus cycle if entire
Musculoskeletal system	Lameness
Nervous system	Seizures or abnormal behaviour
Eyes	Eye problems or abnormal vision
Ears	Ear problems or abnormal hearing
Any other medical problems	Owner to describe

Table 1.3 Skin diseases that usually show age-related onset

Age at onset	Disease
Less than 6 months	Demodicosis Dermatophytosis Juvenile cellulitis Dermatomyositis Impetigo Pituitary dwarfism Histiocytoma Zinc responsive dermatosis Congenital skin diseases
1 to 3 years	Atopic dermatitis Primary seborrhoea Histiocytoma Zinc responsive dermatosis
Middle to old age	Cushings disease Cutaneous neoplasia Cutaneous manifestations of systemic disease

Sex

Most skin disorders do not show sex predispositions. However, diseases of the gonads can have striking skin manifestations such as the symmetrical alopecia seen with Sertoli cell tumours.

Breed

Many skin diseases have hereditary influences and show striking breed predilections (Table 1.4). This information is vital when generating lists of differential diagnoses.

Origin of the animal

It is important to know when the animal was acquired by the owner, and from where. If the animal has been in the owner's possession for all of its life, it is likely that the history of the condition is known from the outset. However, if the animal has only just been obtained and has a pre-existing condition, much of the history will be unknown. The source of the animal is also important. If the animal was obtained as a stray or purchased from a rescue centre, it is more likely to have a parasitic, infectious or nutritional problem. Alternatively, if the animal was obtained from a breeder, it may be possible to gain information about the familial nature of the condition.

Vaccination and worming status

The previous vaccination and worming history of the animal should be recorded as baseline information, and in countries in which heartworm is present, details of the preventative used should be obtained. Although important, this information is not as helpful in generating differential diagnoses for skin conditions as it is for some other body systems. This is because skin conditions associated with viruses and internal parasites are quite rare. Examples would be the rare conditions of footpad hyperkeratosis associated with canine distemper virus infection; dermatoses associated with feline leukaemia virus and feline immunodeficiency virus; facial dermatosis due to feline herpes virus infection; pododermatitis due to penetration of hookworm larvae; pruritus associated with intestinal helminth hypersensitivity; and cutaneous nodules associated with *Dirofilaria immitis* larvae.

Table 1.4 Breed predilection for skin diseases

Breed	Disease
Afghan hound	Hypothyroidism Demodicosis
Airedale	Atopic dermatitis Cyclic flank alopecia
Akita	Pemphigus foliaceus Sebaceous adenitis Uveodermatological syndrome
Alaskan malamute	Demodicosis Hypothyroidism Zinc responsive dermatosis
Basset hound	Atopic dermatitis Malassezia dermatitis Intertrigo
Belgian shepherd dog	Sebaceous adenitis Vitiligo
Boston terrier	Demodicosis Intertrigo Hyperadrenocorticism
Boxer	Atopic dermatitis Demodicosis Cyclic flank alopecia Hyperadrenocorticism Hypothyroidism Chin furunculosis Interdigital furunculosis
Bulldog	Atopic dermatitis Demodicosis Intertrigo Staphylococcal folliculitis Interdigital furunculosis Chin furunculosis Hypothyroidism Malassezia dermatitis
Bullmastiff	Staphylococcal folliculitis and furunculosis
Cairn terrier	Atopic dermatitis
Cavalier King Charles Spaniel	Primary seborrhoea Syringohydromelia (Arnold Chiara syndrome)
Chihuahua	Demodicosis
Chow Chow	Pemphigus foliaceus Atopic dermatitis Colour dilution alopecia Hypothyroidism Adrenal sex hormone alopecia Uveodermatological syndrome
Cocker spaniel	Atopic dermatitis Hypothyroidism Lip fold intertrigo Malassezia dermatitis Otitis externa Primary seborrhoea

Table 1.4 Breed predilection for skin diseases (cont.)

Breed	Disease
Collie	Dermatomyositis Cutaneous lupus Pemphigus erythematosus
Curly coated retriever	Follicular dysplasia
Dachshund	Alopecia areata Demodicosis Colour dilution alopecia Staphylococcal folliculitis Hypothyroidism Hyperadrenocorticism Malassezia dermatitis Sterile nodular panniculitis Pattern baldness
Dalmatian	Atopic dermatitis Demodicosis Staphylococcal folliculitis
Doberman pinscher	Acral lick dermatitis Colour dilution alopecia Demodicosis Drug eruption to potentiated sulphonamides Flank alopecia Hypothyroidism Chin furunculosis Vitiligo Primary seborrhoea
German shepherd dog	Atopic dermatitis German shepherd dog pyoderma Nasal planum lupus Lupoid onychodystrophy Metatarsal fistulae Mucocutaneous pyoderma Pituitary dwarfism Vitiligo
Golden retriever	Acral lick dermatitis Acute moist dermatitis Atopic dermatitis Staphylococcal folliculitis Hypothyroidism Juvenile cellulitis Nasal depigmentation
Great Dane	Acral lick dermatitis Callus Demodicosis Hypothyroidism Chin furunculosis Interdigital furunculosis
Greyhound	Pattern baldness Vasculopathy
Hungarian Vizsla	Sebaceous adenitis
Irish setter	Acral lick dermatitis Atopic dermatitis Colour dilution alopecia Staphylococcal folliculitis and furunculosis Hypothyroidism Primary seborrhoea

Table 1.4 Breed predilection for skin diseases (cont.)

Breed	Disease
Irish water spaniel	Follicular dysplasia
Irish wolfhound	Elbow callus Hypothyroidism
Jack Russell terrier	Atopic dermatitis Demodicosis Dermatophytosis (*Trichophyton erinacei*) Vasculitis
Keeshond	Adrenal sex hormone alopecia
Labrador retriever	Acral lick dermatitis Acute moist dermatitis Atopic dermatitis Nasal hyperkeratosis Staphylococcal folliculitis and furunculosis Malassezia dermatitis
Lhasa apso	Atopic dermatitis Malassezia dermatitis
Newfoundland	Staphylococcal folliculitis and furunculosis Hypothyroidism Pemphigus foliaceus Acute moist dermatitis
Old English sheepdog	Atopic dermatitis Demodicosis Interdigital furunculosis
Pekingese	Intertrigo
Persian cat	Cheyletiellosis Dermatophytosis Intertrigo Matted fur Idiopathic facial dermatosis
Pointer	Demodicosis Exfoliative lupus
Pomeranian	Adrenal sex hormone alopecia
Poodle	Epiphoral staining Hyperadrenocorticism Hypothyroidism Sebaceous adenitis (standard poodle)
Portuguese water dog	Follicular dysplasia
Pug	Atopic dermatitis Intertrigo
Rhodesian Ridgeback	Dermoid sinus
Rottweiler	Staphylococcal folliculitis and furunculosis Vitiligo
Samoyed	Adrenal sex hormone alopecia Sebaceous adenitis Uveodermatological syndrome
Scottish terrier	Atopic dermatitis Demodicosis Staphylococcal folliculitis and furunculosis

Table 1.4 Breed predilection for skin diseases (cont.)

Breed	Disease
Shar pei	Atopic dermatitis Demodicosis Intertrigo Staphylococcal folliculitis Hypothyroidism Mucinosis
Schnauzer	Atopic dermatitis Flank alopecia Hypothyroidism Schnauzer comedone syndrome
Shetland sheepdog	Dermatomyositis Lupus Staphylococcal folliculitis
Shi Tzu	Atopic dermatitis Malassezia dermatitis
Siberian husky	Nasal lupus Eosinophilic granuloma Follicular dysplasia Uveodermatological syndrome Zinc responsive dermatosis
Springer spaniel	Hypothyroidism Lip fold intertrigo Malassezia dermatitis Otitis externa Primary seborrhoea
St. Bernard	Acral lick dermatitis Staphylococcal folliculitis and furunculosis Acute moist dermatitis Lip fold intertrigo
West Highland white terrier	Atopic dermatitis Demodicosis Malassezia dermatitis Primary seborrhoea
Wire haired fox terrier	Atopic dermatitis
Yorkshire terrier	Atopic dermatitis Colour dilution alopecia
Weimeraner	Demodicosis Chin furunculosis
Whippet	Colour dilution alopecia

The type of parasite preventative used by the owner has become more important recently with the introduction of Selamectin, a product capable of providing protection against both internal and external parasites. Hence, if a dog is receiving Selamectin for helminth and heartworm control, it should also be protected against fleas, lice, scabies mites, ear mites, and *Cheyletiella* mites. However, although the use of this product makes it unlikely that the animal would have any of these conditions, the clinician should not rule them out completely on the basis of history alone because resistance to the product is likely to occur at some stage in the future.

What is the owner's main complaint?

It is important to establish from the outset what the client perceives to be the main problem. The most common dermatological problems presented to veterinary surgeons are:

- Pruritus
- Alopecia
- Lumps and swellings
- Otitis.

Less common problems noticed by clients include visible skin lesions such as:

- Excessive scaling
- Papulo-pustular eruptions (rashes).

Rare problems presented by clients include:

- Ulcerative lesions
- Draining tracts or non-healing wounds
- Changes in the animal's skin or coat colour.

Identifying the predominant problem is the first step in generating a differential diagnosis for skin disorders.

How long has the problem been present?

The length of time that a skin problem has been present can have a profound effect on its appearance. Parasitic conditions may spread, hypersensitivities may become more severe, lumps may grow and endocrine alopecias can become more extensive. In addition, the skin tends to react to continued injury by developing certain pathological changes such as lichenification and hyperpigmentation. These chronic changes can mask important diagnostic information such as typical lesion distributions and the presence of primary lesions.

What was the condition like at the beginning?

An accurate description of what the owner first noticed can overcome some of the problems associated with chronic skin problems. It is important to establish what were the initial clinical signs, what lesions were seen and what was their distribution. For example, a dog may present with generalised pruritus. However, on questioning, the owner may reveal that the problem initially only affected the face and feet. As many skin conditions have fairly precise distribution patterns such as this, the information enables the clinician to prioritise the differential diagnosis list.

How has the problem changed over time?

It is important to ask the client how the skin condition has changed since they first noticed it. This can also overcome some of the problems associated with chronicity. For example, a dog may present with pruritus of a few months' duration. However, on careful questioning the owner may reveal that the first lesions seen were symmetrical patches of alopecia and scaling over the trunk. These areas then developed a red rash that the dog started scratching at. In this case, although the owner has presented the dog for pruritus, it is not the predominant problem. The clinician should direct the diagnostic effort towards the cause of the alopecia and scaling.

It is also important to determine the rate and pattern of changes. Skin conditions can:

- Progress rapidly – e.g. parasitic conditions, infectious conditions, auto-immune diseases
- Progress slowly – e.g. allergic diseases, endocrine diseases, skin tumours
- Wax and wane – e.g. allergic diseases
- Change with the seasons – e.g. allergic diseases, seasonal parasites, seasonal alopecia.

Further specific information about pruritus

Pruritus is a sensation in the skin that leads to a desire to scratch. It is the most common problem mentioned by dog owners during a dermatological history and can be manifested as scratching, chewing, licking, rubbing, rolling, or head shaking. In cats, pruritus is less commonly noticed by owners and is usually manifested as either scratching or excessive grooming.

Determining if an animal is pruritic or not is one of the most important questions in a dermatological history. This is because most of the common conditions in veterinary dermatology are pruritic and the clinician is immediately able to develop a list of differential diagnoses. As pruritus can not be accurately assessed on physical examination, it is up to the client to provide more detailed historical information than for the other presenting problems. To extract this information, the clinician should ask questions about three specific aspects of the pruritus:

1. The distribution
2. The severity
3. The onset relative to other skin lesions.

Distribution

When assessing the distribution, the clinician should avoid asking the question 'Where does the animal scratch?' This usually yields incomplete information. Instead, the client should be asked 'Is the animal itchy at any of the following sites?: muzzle; face; around the eyes; ears; neck; back; the sides of the chest and flanks; belly; under the tail; the legs; or the feet.' This type of detailed questioning usually provides much more accurate information than merely asking the client to recall the distribution.

Severity

The clinician should then try to ascertain the severity of the pruritus. This is important for two reasons. First, some diseases are severely pruritic (e.g. canine scabies, acute moist dermatitis, feline eosinophilic plaque) whereas others may be very mildly pruritic (e.g. demodicosis, dermatophytosis). Second, the level of pruritus at initial presentation is used as a starting point to monitor the subsequent response to treatment.

However, assessing the level of pruritus can be particularly difficult because there is no objective scale that the clinician can use. The clinician has to rely on information provided by the client and what is observed in the consulting room. Although there is no perfect way of recording the severity of pruritus, most veterinary dermatologist use one of three systems:

- Basic descriptors such as mild, moderate or severe. Although this is simple to use, the restriction to only three categories means that it is not particularly useful for assessing marginal improvements in the condition.
- A numerical score (usually between 0 and 10). In this system, clients are usually told that 0 is a normal dog and 10 is continuous scratching. The problem with this

Canine itch scale – itch includes scratching, biting, licking, chewing or rubbing

Grade 0:	Normal dog – the dog does not itch more than before the disease began.
Grade 1:	Occasional episodes of itching – small increase in itch compared with before the disease began.
Grade 2:	More frequent episodes of itching, but the itching stops when the dog is sleeping, eating, playing, exercising or is otherwise distracted.
Grade 3:	Regular episodes of itching are seen when the dog is awake. The dog occasionally wakes up because of itching, but the itching stops when the dog is eating, playing, exercising or is otherwise distracted.
Grade 4:	Prolonged episodes of itching are seen when the dog is awake. The dog regularly wakes up because of the itching or itches in its sleep. The itching can also be seen when the dog is eating, playing, exercising or is otherwise distracted.
Grade 5:	Almost continuous itching which does not stop when the dog is distracted, even in the consulting room. The dog needs to be physically restrained from itching.

system is that clients have nothing to compare their animal to, so it is inherently inaccurate. Hence, they tend to over-estimate the severity of pruritus. However, it does provide a starting point against which future comparisons can be made.

- An itch scale based on more complex written descriptors (see Box). Although this allows an owner to choose a level of pruritus based on some objective criteria, it can be difficult to place some animals into a specific category. However, owners are less likely to grossly over-estimate the severity of pruritus with this system than with the other two.

Clinicians should decide for themselves which system they think best suits their needs. This is likely to be based on their relative interest in the discipline, their dermatological caseload and their own style of consulting.

Onset relative to other skin lesions

Determining if the pruritus came before or after the development of other skin lesions is critically important when assessing the history of a particular skin condition. This allows the clinician to determine if they are dealing primarily with a pruritic disease that may have lead to the skin lesions, or if the pruritus is merely a complication of the original disease. For example, if the animal was pruritic and then developed patches of alopecia, it may imply that the hair loss is merely due to self-trauma. In this case, treatment of the cause of the pruritus would also resolve the alopecia. However, if the hair loss preceded the pruritus, it suggests that a complicating factor of the alopecia (such as secondary infection) has caused the pruritus. In this case, treatment of the pruritus would not resolve the original condition. This concept is often summarised by veterinary dermatologists as follows: if an animal has a rash, find out if it is a rash that itches, or an itch that rashes!

Is the condition contagious?

Establishing if a condition is contagious can be very helpful when trying to establish the cause of skin conditions. The client should be asked if there are any other animals or people in the house that have skin lesions. If so, the clinician should determine if the conditions might be related. The skin conditions that most commonly affect other animals and people include fleabite dermatitis, scabies, cheyletiellosis and dermatophytosis.

Is there any familial history?

Owners should be asked if they can provide any medical details about the animal's sire, dam or siblings. This is useful for conditions that are known to have hereditary influences such as atopic dermatitis. Unfortunately, in most cases, owners will not be able to provide this information.

Have any previous treatments been administered?

Animals with skin diseases are often re-presented after being seen by a professional colleague or for a second opinion. Hence, previous treatments may have been prescribed by another veterinary surgeon. Alternatively, treatments may have been administered by the client themselves. There is also a whole range of products that are available in pet shops, pharmacies or health food stores that can be used by clients to treat skin diseases. These include flea control products, shampoos, powders, creams, or nutritional supplements. It is important to establish precisely what drugs or products have been used prior to the animal being presented. It is also important to establish how long these treatments were used for and if any were effective. This information may provide the clinician with clues as to the possible cause of the condition. However, the information should be interpreted with caution because some supposed home remedies are totally ineffective. On the other hand, some veterinary prescribed drugs can appear to be very effective without yielding any diagnostic information. The previous use of glucocorticoids to treat pruritus would be a good example of the latter problem.

What does the animal eat?

Owners often think that their dog or cat's skin disease may be associated with its diet. This is much rarer than is commonly believed. However, the clinician should record precise details of what the animal eats, including meat sources (canned or fresh), dry food (complete, mixer, biscuits), treats or scraps, and nutritional supplements (vitamins, minerals, oils, etc). The owner should also be asked if they have attempted to change the diet in order to influence the skin condition. Skin conditions that may be associated with diet include food intolerance (food allergy), zinc responsive dermatosis and seborrhoea.

Where does the animal live?

Although most skin diseases can occur anywhere in the world, some have very precise geographical distributions. These are usually caused by specific parasites or infectious agents that require particular habitats or climatic conditions. Some are restricted to certain countries whereas others are restricted to certain regions within a country. In other cases, although the condition is not restricted to one area, it may be much more common than in other areas. Hence, clinicians should initially become aware of the conditions that are known to be restricted to, or prevalent in, their own area. This information is best obtained from other clinicians that have been working in the area for some time. Some examples include:

- Fleas – not present in certain parts of the world that are at high altitude or extremely dry
- *Notoedres cati* – restricted to certain regions in certain countries (e.g. parts of the USA, parts of the Mediterranean basin, virtually absent from the UK)
- *Trombicula autumnalis* (harvest mites, berry bugs) – restricted to certain parts of the UK
- *Sarcoptes scabiei* – more common in some locations than in others
- *Cheyletiella* infestation – more common in some locations that in others
- Leishmaniasis – most prevalent in the Mediterranean basin and South America but occurs in parts of the USA and rarely in the UK
- Blastomycosis – only found in the Mississippi region of the USA
- Histoplasmosis – only found in the Mississippi region of the USA
- Coccidiomycosis – only found in the south western regions of the USA
- Cryptococcosis – found in USA and Australia but rarely in UK
- Sporotrichosis – found in USA but extremely rare in UK.

It is likely that the clinician will already know where the animal lives without having to ask the client. However, it is important to ask if the animal has ever lived in, or recently travelled to, another area. This is particularly important now that the quarantine laws have been relaxed in some countries, including the UK.

In addition to knowing the geographical location in which the animal resides, the clinician should obtain details of the animal's home environment. This should include whether it is a rural or urban environment, where the animal sleeps (kennel, kitchen, bedroom, etc.), whether it has free range in the house and whether any environmental changes have been made by the owner. This information can be useful in the investigation of allergic disorders such as atopic dermatitis and contact dermatitis.

General medical history

In the majority of cases of skin disease, the animal is systemically well and will not have any other relevant clinical signs. Conditions that may result in systemic as well as dermatological signs are summarised in Table 1.5. These conditions tend to fall into a number of categories such as endocrine diseases, immune-mediated diseases, severe infections/infestations and neoplasia. Note that this table only includes conditions that have dermatological manifestations and does not list other causes of the clinical signs.

Table 1.5 Possible dermatological conditions associated with systemic signs

Clinical sign	Dermatological condition
Lethargy	Chronic scabies Generalised pustular demodicosis Generalised deep pyoderma Deep and systemic mycoses Pemphigus Lupus Erythema multiforme Toxic epidermal necrolysis Hypothyroidism Hyperadrenocorticism
Weight gain	Hypothyroidism Hyperadrenocorticism
Polyphagia	Hyperadrenocorticism Hyperthyroidism
Polydipsia	Hyperadrenocorticism Hyperthyroidism
Coughing	Systemic mycoses Metastatic neoplasia
Sneezing	Allergic rhinitis Nasal aspergillosis
Vomiting	Food intolerance/allergy Mast cell tumours (gastric ulceration)
Diarrhoea	Food intolerance/allergy
Polyuria	Hyperadrenocorticism
Decreased libido	Stertoli cell tumour Hyperadrenocorticism Hypothyroidism
Altered or ceased oestrus cycle	Ovarian cysts Hyperadrenocorticism
Lameness	Interdigital furunculosis Footpad hyperkeratosis/fissures Pedal neoplasia Systemic lupus erythematosus
Seizures	Cryptococcosis
Head tilt/nystagmus	Otitis interna
Ocular discharge	Allergic conjunctivitis
Aural discharge/abnormal hearing	Otitis externa or media

2

Physical examination

After obtaining a history, a complete dermatological and general physical examination should be performed. Although it is tempting to focus immediately on the visually striking skin lesions pointed out by the client, this should be avoided. Instead, clinicians should develop a logical system of examination of both the skin and internal organs. Once established, this routine should be applied to every case. This approach ensures that lesions that have not been noticed by the client are not missed. As with history taking, it is the clinician's own preference as to whether the dermatological examination or general examination is performed first.

Examination of the skin

There are three major aims when performing a dermatological examination:

1. To detect the presence of grossly visible parasites
2. To identify any lesions that are present
3. To determine the distribution of the lesions.

It is important to establish a routine when examining the skin so that the whole body area is covered. The clinician should first observe the entire animal from a distance to judge the appearance of its coat. Then, the skin and coat should be examined in detail under good lighting. Usually, the examination starts at the head and proceeds distally. The clinician should examine the muzzle, lips, chin, nasal planum, face, periocular region, medial and lateral aspects of the pinnae, ear canals, dorsal and ventral neck, dorsum, lateral chest, tail, perineum, axillae, ventral abdomen, legs, interdigital areas and pads.

Whilst examining these body regions, the clinician should feel the quality of the coat and determine if it is dry, greasy or easily epilated. The clinician should also look for grossly visible ectoparasites such as fleas, lice or *Trombicula* larvae, or clues to their presence such as flea dirt or lice eggs. The skin surface should then be carefully examined by running the fingers against the lay of the hair. If necessary, the hair coat may need to be clipped in order to visualise subtle lesions that would otherwise be hidden. It may also be occasionally helpful to use a magnifying glass to assess very small lesions.

An important technique that can be used to help assess the degree of pruritus in dogs is digital stimulation. As each body region is examined, the surface of the skin should be firmly scratched with one or more fingernails. If the skin is pruritic, this may trigger a reflex scratching action of the dog's hind leg. If this occurs when the margins of the pinnae are scratched, it is known as the pinnal-pedal scratch reflex and is suggestive of canine scabies. This technique can be used to determine if the skin is generally pruritic or if the pruritus is restricted to particular lesions. If the reflex is elicited when a single lesion is stimulated, but stops when an adjacent patch of normal skin is scratched, it indicates that the pruritus is resulting from the lesions themselves. This distinction becomes critical when dealing with staphylococcal pyoderma, a secondary infection that can be superimposed on many other skin conditions. With this technique, the clinician may be able to predict if the pruritus will resolve following treatment of the bacterial lesions alone.

In addition to observing and touching the skin, the clinician should also smell the skin. Many disorders of the skin or ears lead to changes in the animal's body odour. Disorders known to produce characteristic odours include canine scabies (mousy odour), Malassezia otitis (yeasty odour), anal sac problems (musky odour), seborrhoea oleosa (rancid fat odour) and anaerobic infections and abscesses (putrid, necrotic odour).

As the examination progresses, the clinician must characterise and record any skin lesions that are observed. For this to be meaningful, clinicians must have a complete understanding of the various types of lesions that can occur on the skin and their diagnostic significance.

Skin lesions

The skin can only respond to injury in a limited number of ways. These pathological changes (or lesions) represent a form of visual language that can communicate to the clinician what type of disease process is occurring. Hence, in order to be able to diagnose skin diseases, clinicians must learn to understand this language and appreciate what the skin is trying to say. However, clinicians should be aware that a single type of lesion is rarely pathognomonic for a particular disease, in the same way that a single word does not convey as much meaning as a sentence. The information derived from all the lesions must therefore be assimilated and processed, allowing the clinician to determine what major patterns of changes are present.

Skin lesions can be classified in a number ways. Many veterinary dermatologists like to describe lesions as either primary or secondary. Primary lesions occur as a direct result of the disease process whereas secondary lesions result from progression of the disease or self-trauma. Primary lesions therefore provide more specific diagnostic information. However, some skin lesions can be either primary or secondary depending on the disease concerned so this classification can, at times, be confusing.

In this book, skin lesions will be classified into categories based on how they actually appear to the clinician during a dermatological examination. These categories are:

- loss of hair
- changes in skin colour
- rashes
- excessive scaling
- changes in skin thickness
- draining tracts
- defects in skin integrity
- abnormal components on the skin surface
- lumps and swellings.

Loss of hair

Loss of hair is a common presenting sign in both dogs and cats. It may occur spontaneously or be secondary to pruritus. Spontaneous alopecia is a cardinal sign of disorders affecting the hair follicle such as follicular infections and endocrine diseases. If secondary to pruritus, the hair is removed by the animal itself by scratching, rubbing, biting, chewing or excessive grooming. Terms used to describe hair loss include:

- Alopecia – loss of hair in any amount up to complete baldness.
  Alopecia may be partial, in which the hair density is merely reduced, or total, in which the area of affected skin is devoid of hair. In animals, alopecia usually occurs as one of four patterns:
  - Focal – a single, small patch of alopecia
  - Multi-focal – multiple, small, circular patches of alopecia giving the coat a moth-eaten appearance
  - Regional – alopecia affecting a region of the body such as one leg
  - Symmetrical – alopecia that has the same distribution on one side of the body as the other.
- Hypotrichosis – less than the normal amount of hair. This term is rarely used in veterinary dermatology and normally describes a congenital deficiency of hair
- Defluxion/effluvium – a sudden, widespread loss of hair
- Easy epilation – the ability to easily remove excessive quantities of hair with little resistance during physical examination. This implies that many hairs are in the resting (telogen) phase. This can be physiological (due to shedding) or pathological. When an animal is shedding, it is not possible to remove enough hair to leave a patch of alopecia. Hence, regardless of the amount of hair being removed, the owner can be reassured that nothing is wrong. On the other hand, if the epilation leads to the development of a bald patch, there is an underlying abnormality that is leading to spontaneous hair loss
- Scar – the abnormal skin at the site of a healed wound. Scars are usually characterised by a lack of hair and pigment, are slightly raised and may have a shiny surface. Linear scars are usually hidden by the hair coat in animals. More extensive scars are visible as large patches of alopecia. Because scars lack hair follicles, the alopecia is irreversible.

Changes in skin colour

The normal skin of dogs and cats is usually a whitish-grey colour, even if the animal has a black coat. In animals with black and white or tricolour coats, some areas of skin may be naturally pigmented but this is usually a darkish-grey colour rather than black. Some white coated breeds such as English Bull Terriers and West Highland White Terriers have pale-pink coloured skin that can become more intense when the dog is excited. Many skin conditions lead to a change in colour of the skin. These colour changes include:

- Erythema – skin that is redder than normal, implying that the skin is inflamed.
  Erythema occurs most commonly in allergic, parasitic, infectious and immune-mediated skin diseases. Erythroderma refers to generalised erythema.
- Hyperpigmentation – skin that is darker than normal.
  Excessive pigment in the epidermis leads to black coloured skin. This occurs most commonly as a chronic change in allergic diseases and Malassezia dermatitis, and as a hormonal effect in endocrine disorders.
  Excessive pigment in the dermis leads to blue-grey coloured skin. This occurs most commonly in demodicosis.
- Hypopigmentation – skin or hair that is paler than normal.
  Loss of pigment from the skin or hair may result from hereditary, autoimmune, nutritional, neoplastic and idiopathic diseases.

- Macules – circular, flat areas of abnormally coloured skin, up to 1 cm in diameter.
    - Erythematous macules are commonly caused by staphylococcal pyoderma and fleabite hypersensitivity. They are also seen with fly bites, contact dermatitis, and erythema multiforme.
    - Haemorrhagic macules (ecchymoses) may be seen with vasculitis and coagulopathies.
    - Hyperpigmented macules are commonly seen with staphylococcal pyoderma at the sites of resolving lesions and on the scrotum, prepuce and anus associated with testicular tumours.
    - Hypopigmented macules may be seen with vitiligo and idiopathic depigmentation.

Rashes

A rash is a collection of skin lesions usually comprised of erythematous macules, papules and pustules (maculo-papulo-pustular eruption).

- Erythematous macules – defined above.
- Papules – small, red, circular pimples less than 1 cm in diameter.
  Papules are most commonly seen with staphylococcal pyoderma, fleabite hypersensitivity, scabies, atopic dermatitis, fly bite hypersensitivity and contact dermatitis. Clinicians should be extremely careful in distinguishing a papular eruption from erythema. Erythema is a diffuse area of inflamed skin whereas a papular eruption will appear mottled with areas of normal skin interspersed amongst the papules. However, with severe papular eruptions, the lesions can become contiguous so the clinician needs to look carefully at the edge of the dense patch of inflammation to see the individual lesions.
- Pustules – red, circular spots containing a central, yellow sac of pus.
  Pustules are commonly seen with staphylococcal pyoderma but may also occur with demodicosis, pemphigus and some rare sterile pustular disorders.
- Crusted papules and pustules – papules and pustules covered in a small crust (scab). These lesions are commonly seen alongside papules and pustules and imply that pus or exudate has come onto the surface and dried. Crusted papules are commonly seen in miliary dermatitis in cats. Crusted pustules are seen in all pustular diseases due to the fragile nature of canine and feline pustules.
- The staphylococcal ring – a specific type of lesion that is commonly seen in staphylococcal pyoderma. The lesion comprises a central, circular area of alopecia (that may or may not be hyperpigmented) surrounded by a rim of erythema with a ring of peripheral scaling (an epidermal collarette).

Excessive scaling

In normal skin, the superficial layers of the stratum corneum (corneocytes) are being continuously desquamated into the environment. These cells are microscopic so are not visible on the skin surface or in the coat. However, in some disease states, this process is disrupted leading to visible skin lesions.

- Scale – grossly visible accumulations of corneocytes (dandruff). Excessive scale may form as a result of any disease that disrupts the normal process of cornification.
- Seborrhoea – a descriptive, clinical term referring to any skin condition characterised by excessive scaling or greasiness. Seborrhoea sicca is used to describe the

appearance of dry and scaly skin. Seborrhoea oleosa is used to describe the appearance of excessively greasy skin.

- Epidermal collarette – a circular ring of scale. Epidermal collarettes are formed when a focal point of infection spreads outwards as an enlarging circle, causing the stratum corneum to lift upwards. The end result is a circular patch of alopecia surrounded by a rim of scale. This extremely common lesion is invariably associated with staphylococcal pyoderma and may be seen alone or in conjunction with papules and pustules. An epidermal collarette often surrounds a staphylococcal ring.
- Exfoliation – the shedding of very large sheets of scale over large areas of the body.
- Hyperkeratosis – a pathological term meaning thickening of the stratum corneum. On the skin surface, hyperkeratosis is only seen histologically. It would be manifested clinically as excessive visible scale. Clinically, the term hyperkeratosis should only be used for pathological accumulations of thick, adherent keratin on the nasal planum or the footpads.
- Comedone – an accumulation of keratin within a hair follicle (blackhead). Comedones occur with diseases that affect hair follicles such as demodicosis, endocrine diseases and cornification defects.
- Follicular cast – an adherent sheath of scale surrounding a hair shaft above the skin surface. This lesion results from follicular hyperkeratosis and is usually found in association with cornification defects.

Changes in skin thickness

Some conditions lead to a change in skin thickness. Increased skin thickness can be caused by thickening of the epidermis or dermis, or infiltration of the skin with inflammatory cells. Decreased skin thickness occurs due to a combined thinning of the epidermis and dermis.

- Lichenification – a marked thickening of the skin due to a dramatic increase in thickness of the epidermis. Lichenification leads to an exaggeration of the visible skin markings so that the skin looks like that of an elephant. It is an attempt by the body to protect itself from further injury by forming a thicker defensive barrier. Lichenification often occurs in combination with hyperpigmentation which is another defensive mechanism. Lichenification most commonly occurs with chronic pruritic skin diseases.
- Plaque – a localised patch of thickened skin with a flat surface. Plaques are either caused by thickening of the epidermis or by infiltration of inflammatory or neoplastic cells. The most common example is feline eosinophilic plaques.
- Callus – a thickened, rough, alopecic, lichenified plaque that forms over pressure points
- Myxoedema – a puffy, thickening of the skin caused by excessive quantities of mucin in the dermis. This is a feature that can be seen with hypothyroidism and in the Chinese Shar Pei breed of dog.
- Cutaneous atrophy – skin that is visibly thinner than normal. This may be appreciated because the cutaneous blood vessels become more prominent or the skin may become hypotonic. Hypotonic skin is less elastic and is easily wrinkled. Cutaneous atrophy may be seen with hyperadrenocorticism and following the excessive local use of glucocorticoids (topical application of creams or subcutaneous injections).

Draining tracts

- Furunculosis – rupture of hair follicles beneath the skin surface. Furunculosis normally occurs due to bacterial infection or demodicosis and results in sinus tracts.
- Sinus – an opening between the underlying dermis or subcutis and the skin surface, through which exudate is normally discharging. Sinuses are normally associated with deep infections.

Defects in skin integrity

The most common example of a defect in skin integrity is a wound. A wound is a traumatically or surgically induced breach in skin continuity. The management of wounds will not be covered in this text and readers should seek further information from surgical textbooks. Other defects in skin integrity that can be caused by dermatological problems include:

- Erosion – a shallow defect in which the epidermis is missing but the basement membrane is intact.
- Ulcer – a deeper defect in which the epidermis is absent exposing the underlying dermis. In dogs and cats, the epidermis is so thin that virtually all erosions become ulcers. Ulcers can be caused by diseases that damage the epidermis or underlying dermis (autoimmune and immune-mediated diseases, deep infections, severe self-trauma, neoplasia, burns).
- Vesicle – a blister less than 1 cm in diameter. Blisters form due to lack of cohesion between the epidermis and dermis. The resulting space becomes filled with tissue fluid. In dogs and cats, the epidermis is so thin that vesicles on the skin are virtually never seen. Diseases that would cause cutaneous blisters in humans cause ulcers in dogs and cats. However, vesicles can rarely be seen in the oral cavity of dogs and cats where the epithelium is thicker. Blistering disorders are caused by autoimmune or congenital diseases of the epidermis or dermo-epidermal junction.
- Bulla – a large blister greater than 1 cm in diameter, virtually never seen in dogs and cats.
- Excoriation – a defect (scratch) in the skin caused by trauma (thorns, barbed wire etc.) or self-trauma. Excoriations often have a linear configuration and may be grouped in parallel rows that correspond to the animal's claws. Excoriations due to self-trauma occur with pruritic diseases.
- Fissure – a spontaneously occurring linear split in the epidermis that exposes the underlying dermis. Fissures usually occur with diseases that affect the nasal planum or footpads.

Abnormal components on the skin surface

- Exudate – a mixture of body fluids and cells that comes out onto the skin surface. Exudates may contain pus, serum and/or blood and are usually associated with infections or ulcerative diseases.
- Crust – an adherent mixture of stratum corneum and other components such as serum, blood or pus. Crusts form when exudates or blood dry on the skin surface.
- Hyperhidrosis – excessive quantities of sweat on the skin surface. Hyperhidrosis may be seen with inflammatory skin diseases, especially atopic dermatitis.
- Calcinosis cutis – deposition of calcium into the dermis which may penetrate through to the exterior exposing white, chalky pieces of grit-like material. Calcinosis cutis can be seen with hyperadrenocorticism.

Lumps and swellings

The presence of a cutaneous lump or swelling is one of the most common reasons for dogs and cats to be presented to veterinary surgeons. For obvious reasons, lumps are a source of great anxiety for owners and the clinician should try and determine as quickly as possible what has caused the lump to occur.

- Abscess – a cavity in the skin filled with pus. Abscesses are invariably caused by infections, although sterile abscesses can rarely form at the sites of injections.
- Haematoma – a cavity in the skin filled with blood. Haematomas are usually caused by trauma.
- Wheal – a circumscribed, circular, raised area of skin caused by oedema in the dermis. Wheals are seen in urticaria, an immediate hypersensitivity reaction. Wheals are also produced at the site of positive skin test reactions when animals are allergy tested.
- Cyst – an epithelial lined cavity filled with skin components such as keratin, corneocytes, hair or sebum.
- Nodule – a firm, solid cutaneous swelling up to 1 cm in diameter caused by infiltration of inflammatory or neoplastic cells.
- Tumour – a solid mass of tissue caused by infiltration or expansion of neoplastic cells.

Lesion distribution

Whilst assessing the type of lesions, the clinician must also record their distribution. The distribution of skin lesions is one of the most important clinical features used to prioritise the differential diagnosis list because many dermatoses have characteristic distribution patterns.

There are two ways in which this information can be recorded. Most veterinary surgeons write down details of the distribution as the examination progresses. However, a more useful method used by the majority of veterinary dermatologists is to record the information on a lesion distribution diagram. An example suitable for photocopying and placing in the animal's record is shown in Figure 2.1.

In addition to being quick and easy to perform, this method provides a visual reference that is useful to refer back to when monitoring progress. It is also valuable when cases are seen by more than one clinician during the course of treatment. An example of a completed lesion distribution diagram is shown in Figure 2.2.

The distribution of lesions provides important clues as to the possible diagnosis. Some typical distribution patterns include:

- Regional – only one part of the body is affected such as a leg or the nasal planum.
- Multi-regional – a number of different body sites are affected such as the face, ears, legs and ventrum.
- Generalised – virtually the whole skin surface is affected.
- Symmetrical – the lesions are the same on one side of the body as on the other. This is usually seen with parasitic, allergic, autoimmune, endocrine or systemic diseases.
- Non-symmetrical – lesions on one side of the body are not mirrored on the other. This is usually seen with infectious or neoplastic diseases.

Details on the distribution of lesions in various skin diseases are provided in Chapters 4–12.

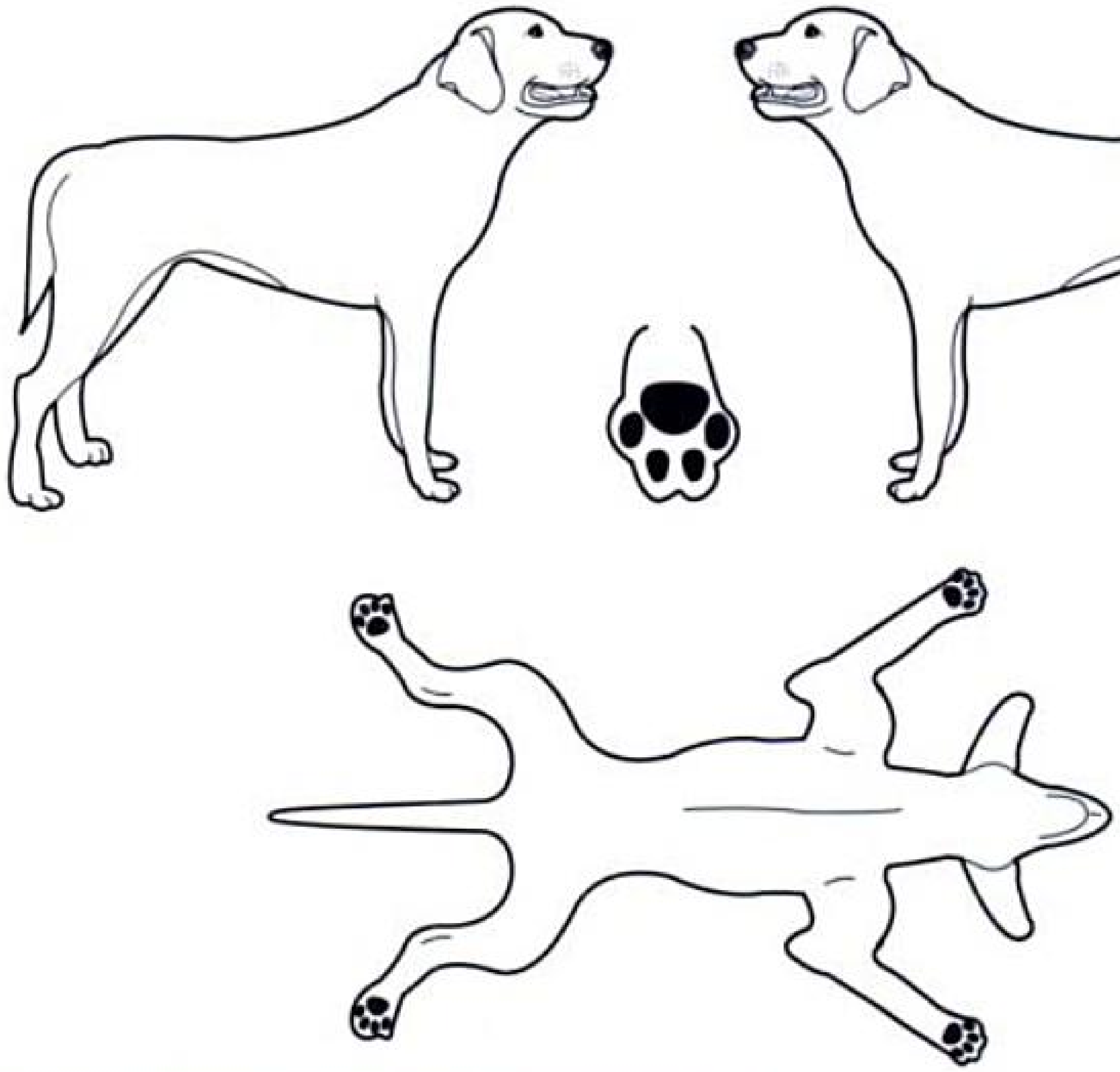


Figure 2.1 Diagram for recording the type and distribution of skin lesions.

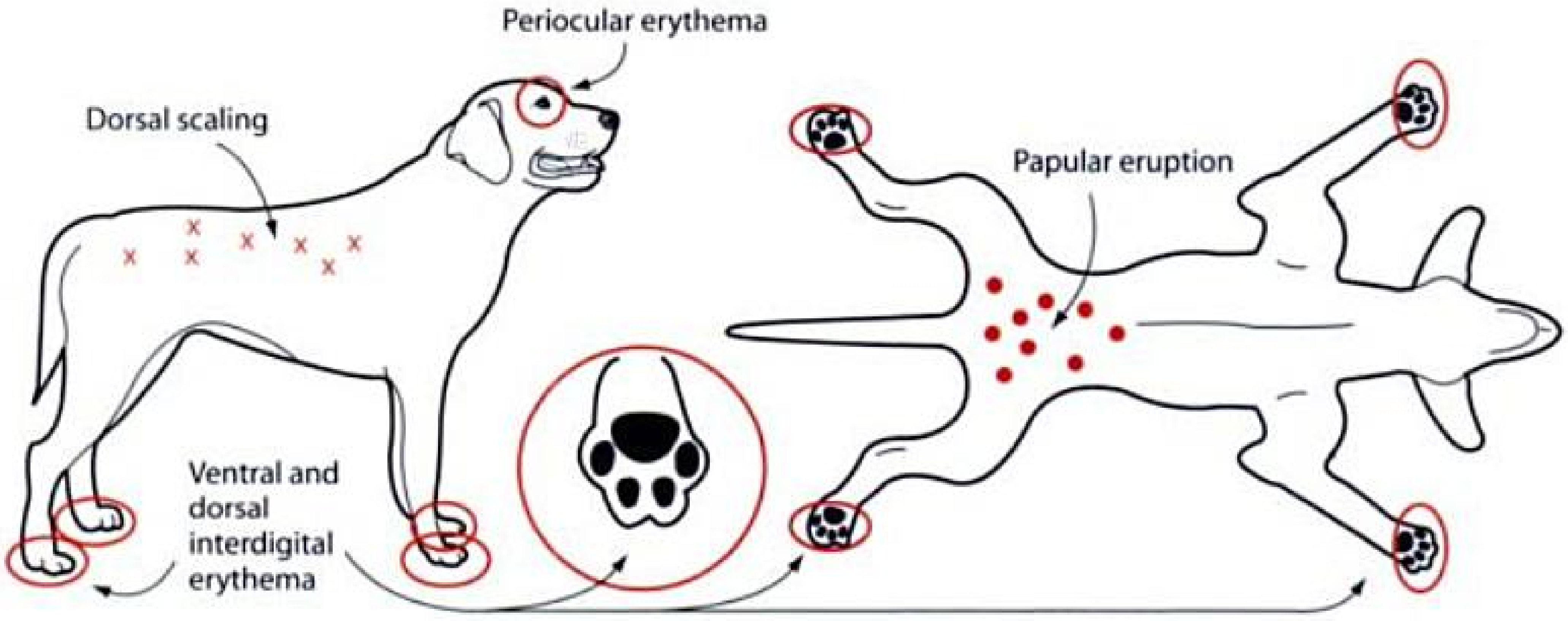


Figure 2.2 Typical lesion distribution diagram.

3

Dermatological problems

After obtaining a detailed history and completing a physical examination, the clinician must combine the information to determine what is the predominant problem(s). Figure 3.1 shows an algorithm that can be used to decide what the primary problem is. The key to the problem-oriented approach is to realise that the predominant problem will usually be the one for which the client has actually presented the animal. Hence, although the history and physical examination may reveal a dog that is pruritic with patches of alopecia, scaling and areas of hyperpigmentation, the owner will usually present the animal for pruritus. This problem should be investigated first because it is likely that the other problems are related. If the other problems persisted after the cause of the pruritus had been eliminated, they would be investigated subsequently.

In some cases, an animal may genuinely have two or more problems that are clearly unrelated. An example would be an itchy dog that also had a lump. In this case a separate list of differential diagnoses should be drawn up for each problem and they should be investigated at the same time.

If the clinician is unsure as to whether two problems are related, it is always safer to draw up differential diagnoses for each problem separately. The two lists can then be scrutinised for conditions that appear on both lists, as these should receive priority in the initial investigation.

The following Chapters (4 to 12) are devoted to the problems identified in Figure 3.1. Each chapter provides:

- A list of differential diagnoses for each problem
- The key clinical features of each disease, including lesion distribution
- A list of the diagnostic tests that can be used to investigate each condition
- A general diagnostic approach for each problem.

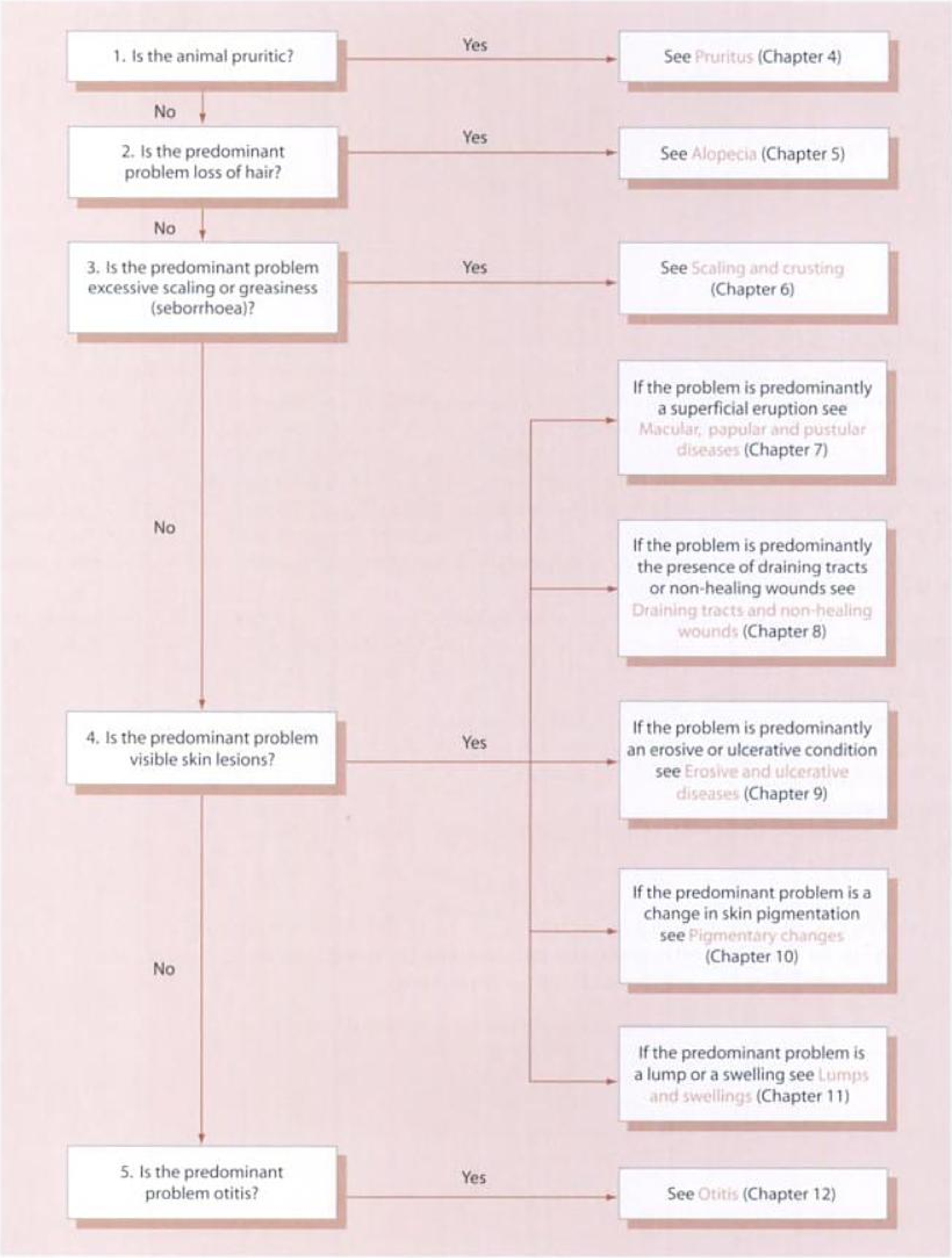

1. Is the animal pruritic?
Yes
See Pruritus (Chapter 4)
No
2. Is the predominant problem loss of hair?
Yes
See Alopecia (Chapter 5)
No
3. Is the predominant problem excessive scaling or greasiness (seborrhoea)?
Yes
See Scaling and crusting (Chapter 6)
No
If the problem is predominantly a superficial eruption see Macular, papular and pustular diseases (Chapter 7)
If the problem is predominantly the presence of draining tracts or non-healing wounds see Draining tracts and non-healing wounds (Chapter 8)
4. Is the predominant problem visible skin lesions?
Yes
If the problem is predominantly an erosive or ulcerative condition see Erosive and ulcerative diseases (Chapter 9)
If the predominant problem is a change in skin pigmentation see Pigmentary changes (Chapter 10)
No
If the predominant problem is a lump or a swelling see Lumps and swellings (Chapter 11)
5. Is the predominant problem otitis?
Yes
See Otitis (Chapter 12)

Figure 3.1 Algorithm to determine the predominant dermatological problem. This information is derived from both the history and physical examination.

4

Pruritus

Differential diagnosis

Pruritus is the most common presenting dermatological problem in dogs and cats. In the majority of cases, pruritus is caused by either an ectoparasite, an infectious agent or an allergy. Table 4.1 shows the major differential diagnoses for pruritus in dogs and cats. Most of the diseases can be seen in both species with the exception of *Sarcoptes scabiei* (usually only seen in dogs), *Notoedres cati* (only seen in cats) and staphylococcal pyoderma (rarely seen in cats).

In dogs, the clinical signs of these diseases are fairly characteristic (see below). Hence, the lesions and their distribution provide important clues when prioritising the differential diagnoses. However, in cats, the situation is much less clear cut. Cats with pruritus tend to present with one of the following four cutaneous reaction patterns:

- Miliary dermatitis
- Symmetrical (self-induced) alopecia
- Head and neck pruritus
- The eosinophilic granuloma complex

Table 4.1 Differential diagnoses for pruritus

Aetiological category	Specific disease
Ectoparasites	**Flea bite hypersensitivity (D and C)**
	Pediculosis (D and C)
	Cheyletiella infestation (D and C)
	Sarcoptes scabiei infestation (D)
	Demodicosis (D and C)
	Trombicula autumnalis infestation (D and C)
	Otodectes cynotis infestation (D and C)
	Insect bite hypersensitivity (D and C)
	Notoedres cati infestation (C)
Infectious agents	**Staphylococcal pyoderma (D)**
	Malassezia dermatitis (D and C)
	Dermatophytosis (D and C)
Allergies	**Atopic dermatitis (D and C)**
	Food allergy (D and C)
	Contact dermatitis (D and C)
Miscellaneous	Epitheliotropic lymphoma (D and C)

Those in bold would be most common in general practice. D = those diseases seen in dogs, C = those diseases seen in cats.

These reaction patterns are not diagnoses. They are clinical presentations that can be caused by any of the diseases listed in Table 4.1 and are described later in this chapter.

Clinical features of pruritic skin diseases

Flea Bite Hypersensitivity

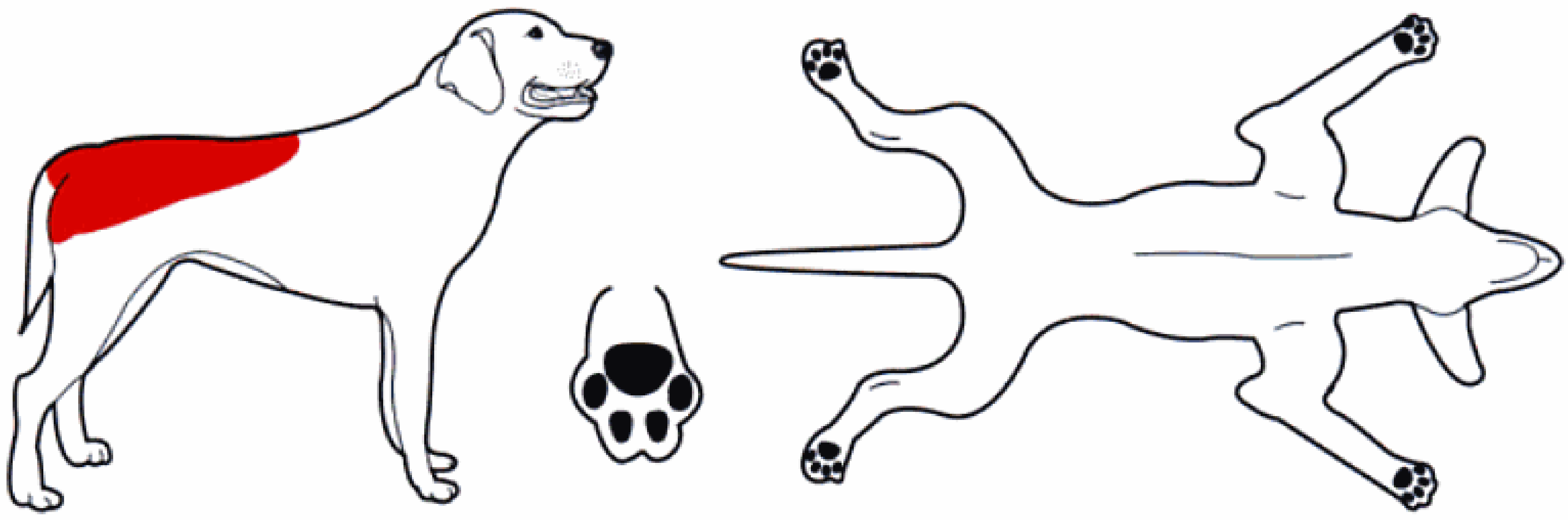
Aetiology: Infestation with fleas leading to an allergic reaction to antigens in flea saliva.

Predominant lesions:

Acute: Erythematous macules, papules, crusted papules, hot spots.
Chronic: Self-induced alopecia, lichenification, hyperpigmentation.

Lesion distribution:

Usually dorsal lumbo-sacral region but may generalise.



Appropriate diagnostic tests:

Direct examination for fleas or flea dirt
Coat brushing
Detection of flea antigen specific-IgE by intradermal testing or ELISA
Response to treatment

Pediculosis

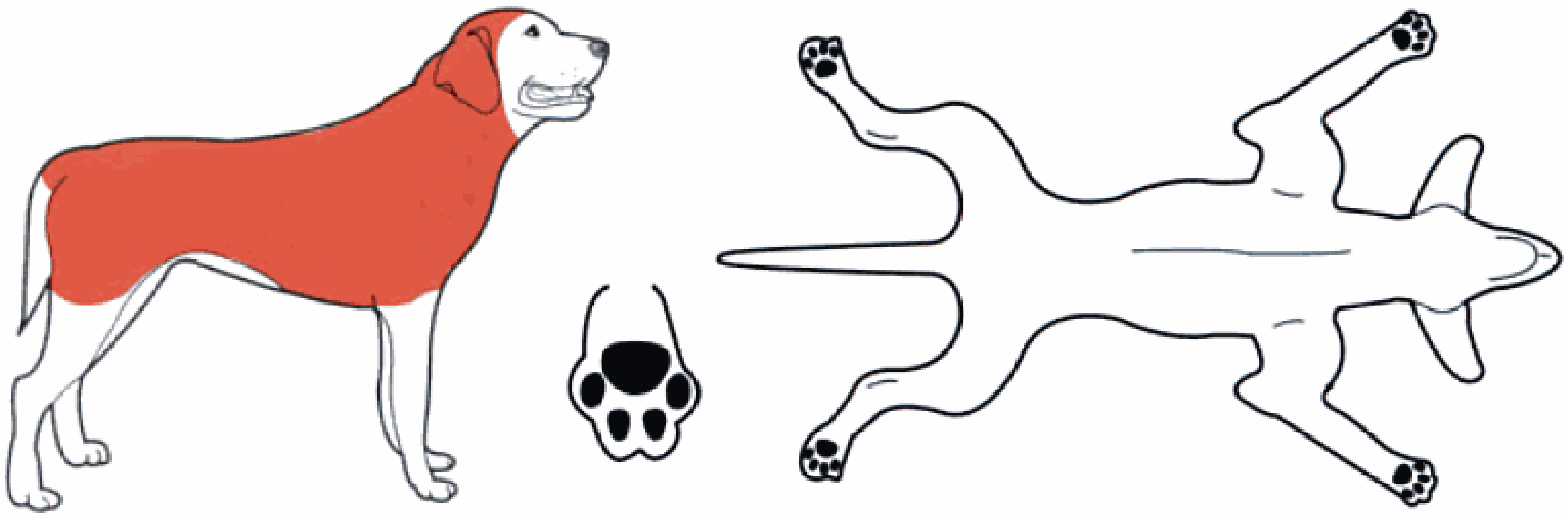
Aetiology: Infestation with lice.

Predominant lesions:

None or mild scaling and excoriations.

Lesion distribution:

Usually on the trunk and head.



Appropriate diagnostic tests:

Direct examination for lice
Coat brushing
Unstained adhesive tape strip

Cheyletiellosis

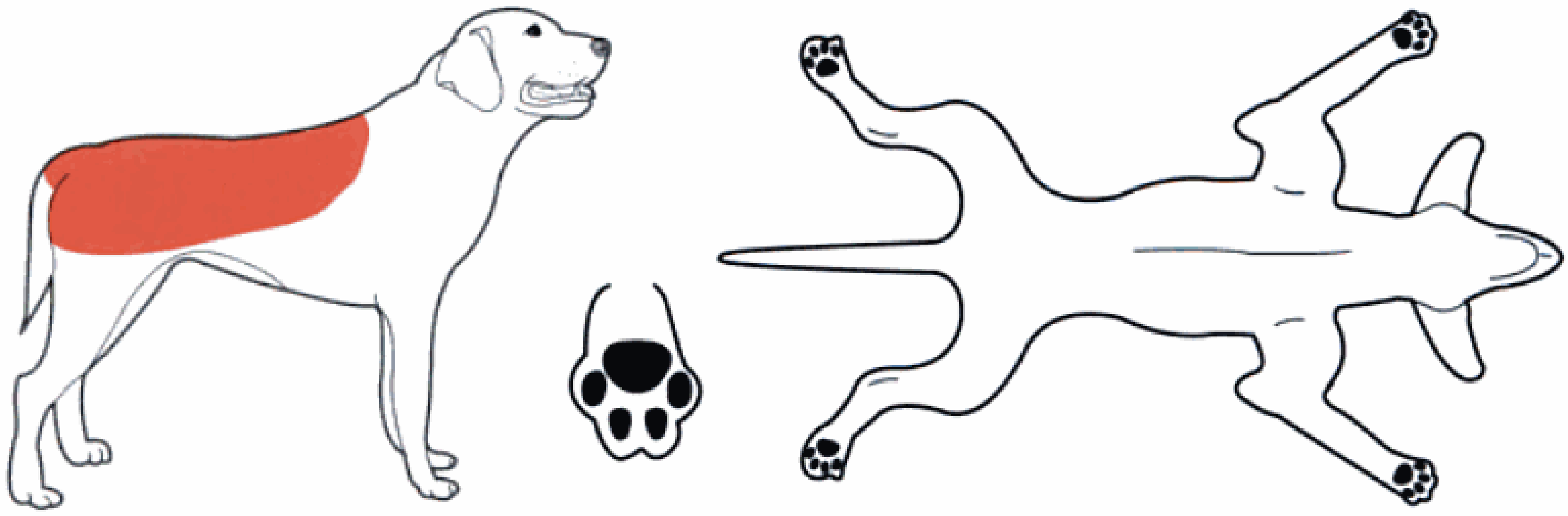
Aetiology: Infestation with Cheyletiella mites.

Predominant lesions:

Excessive scaling.

Lesion distribution:

Over the dorsum.



Appropriate diagnostic tests:

Coat brushing
Unstained adhesive tape strip
Skin scraping
Response to treatment

Scabies (sarcoptic mange)

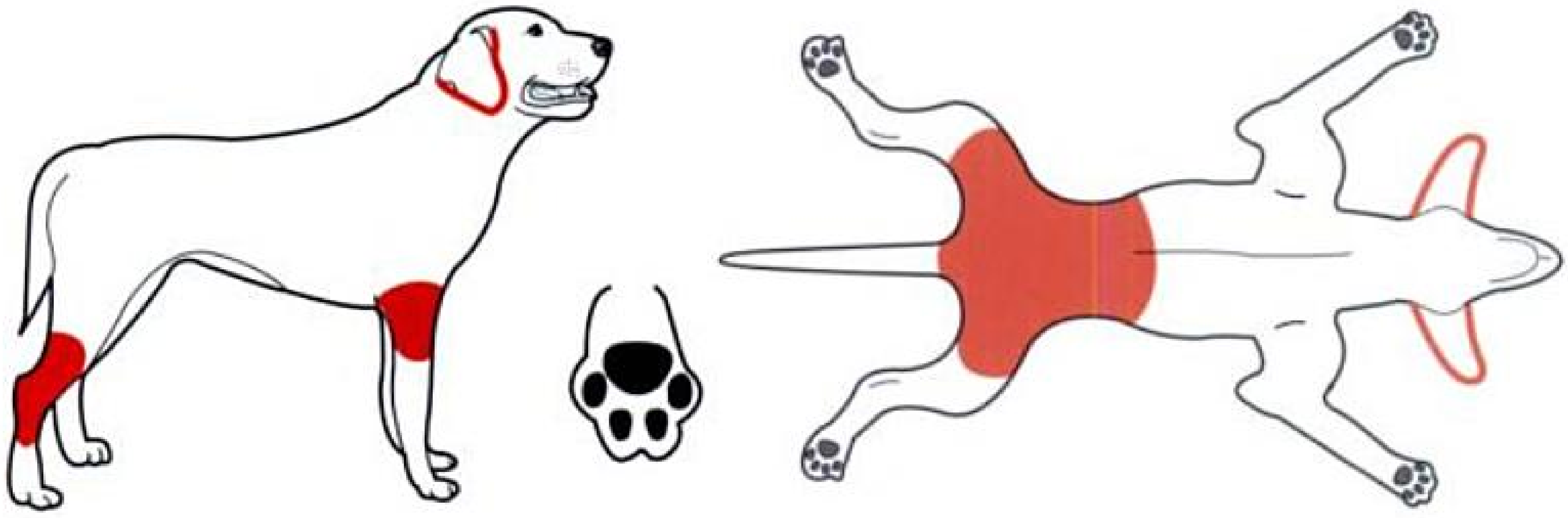
Aetiology: Infestation with *Sarcoptes scabiei* mites.

Predominant lesions:

Papular eruption, erythema, scaling, excoriations.

Lesion distribution:

Margins of the pinnae, elbows, hocks, ventral abdomen.



Appropriate diagnostic tests:

Skin scrapings
Detection of scabies-specific IgG by ELISA
Response to treatment

Demodicosis (demodectic mange)

Although demodicosis should be considered in the differential diagnosis of pruritus, it most commonly presents as alopecia (see Chapter 5 for further details).

Trombicula infestation

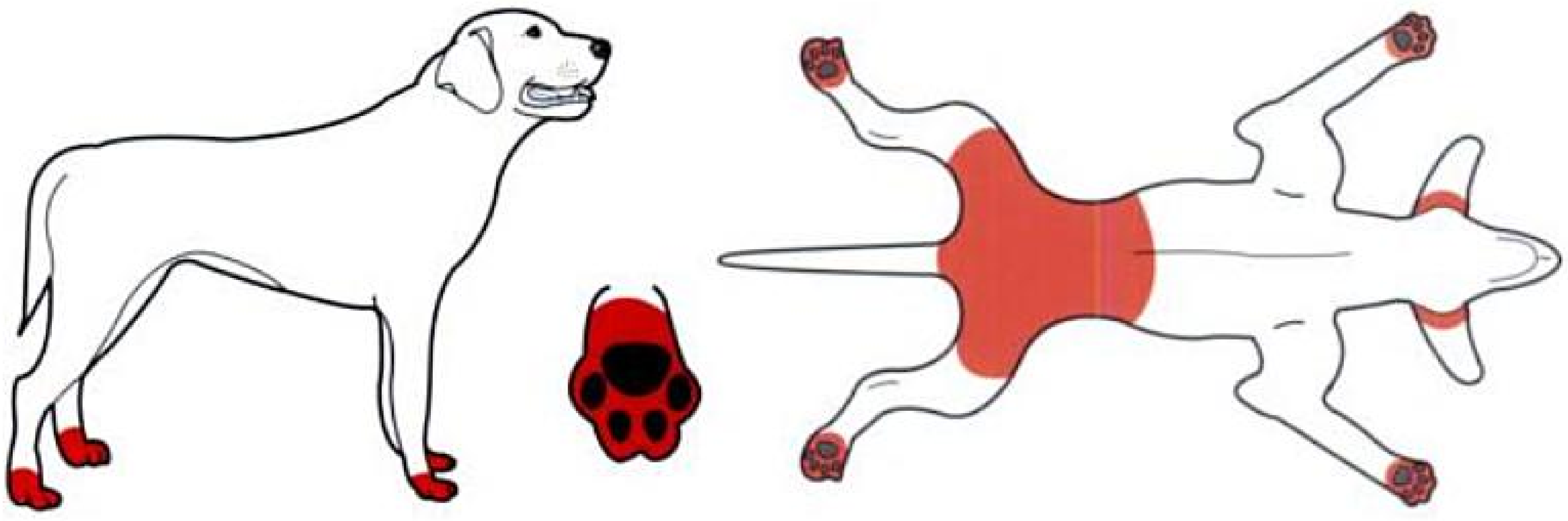
Aetiology: Parasitism by the larvae of Trombicula mites.

Predominant lesions:

Papular eruption.

Lesion distribution:

Feet, ventral abdomen, external ear canals and pinnae.



Appropriate diagnostic tests:

Direct examination for parasites
Unstained adhesive tape strip
Skin scraping

Otodectes cynotis infestation

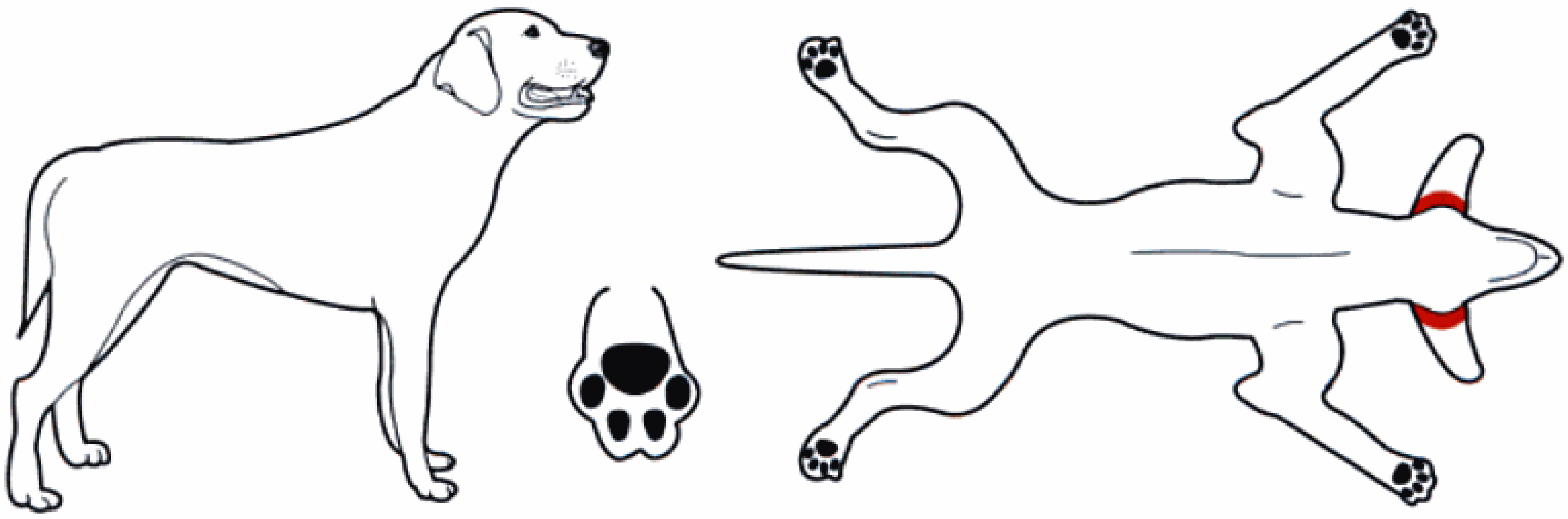
Aetiology: Parasitism of the ear canals with ear mites.

Predominant lesions:

Browny-black, dry, crusty exudate in the ear canals.

Lesion distribution:

Usually external ear canals. Rarely spreads to the body.



Appropriate diagnostic tests:

Otoscopic examination
Examination of exudate for parasites
Skin scraping if lesions on body

Notoedric mange

Aetiology: Infestation with *Notoedres cati* mites.

Predominant lesions:

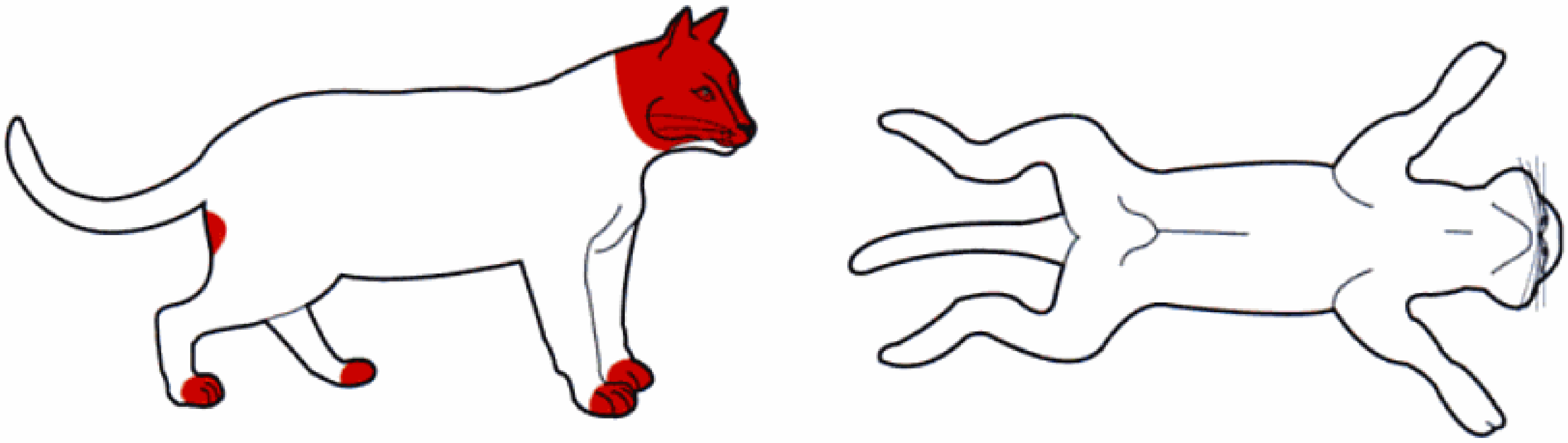
Papules, excessive scaling, crusts, excoriations.

Lesion distribution:

Pinnae, face, neck, feet, perineum.

Appropriate diagnostic tests:

Skin scraping
Response to treatment



Insect bite hypersensitivity

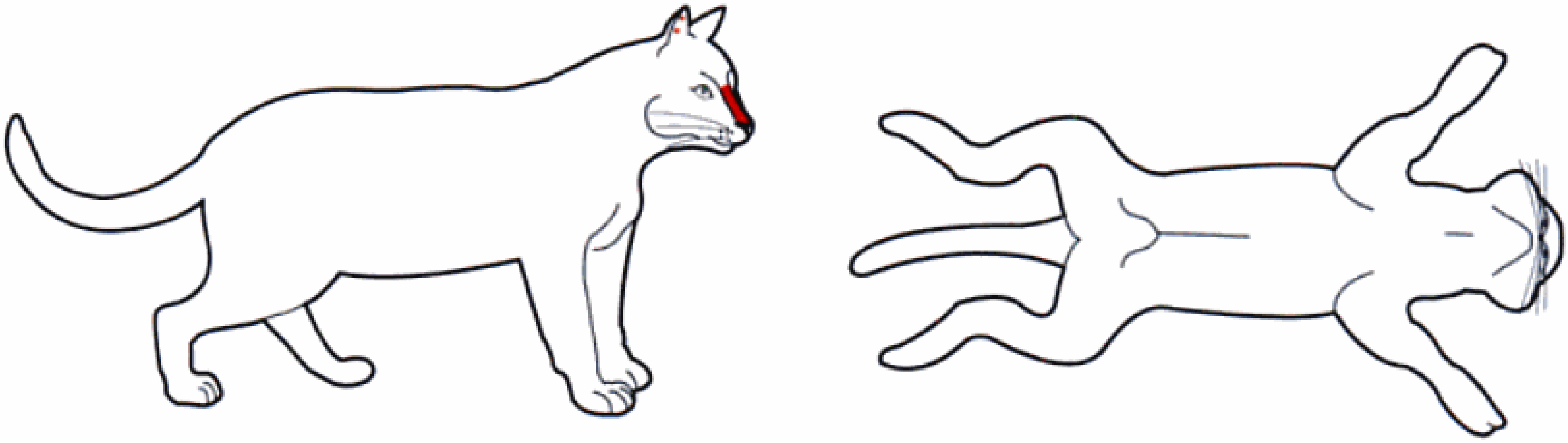
Aetiology: Allergic reaction to biting flies such as mosquitoes, culicoides, blackflies.

Predominant lesions:

Papules, crusted papules, ulcers, eosinophilic furunculosis.

Lesion distribution:

Bridge of nose, ear tips.



Appropriate diagnostic tests:

Cytology
Response to treatment

Superficial pyoderma (superficial folliculitis, staphylococcal pyoderma)

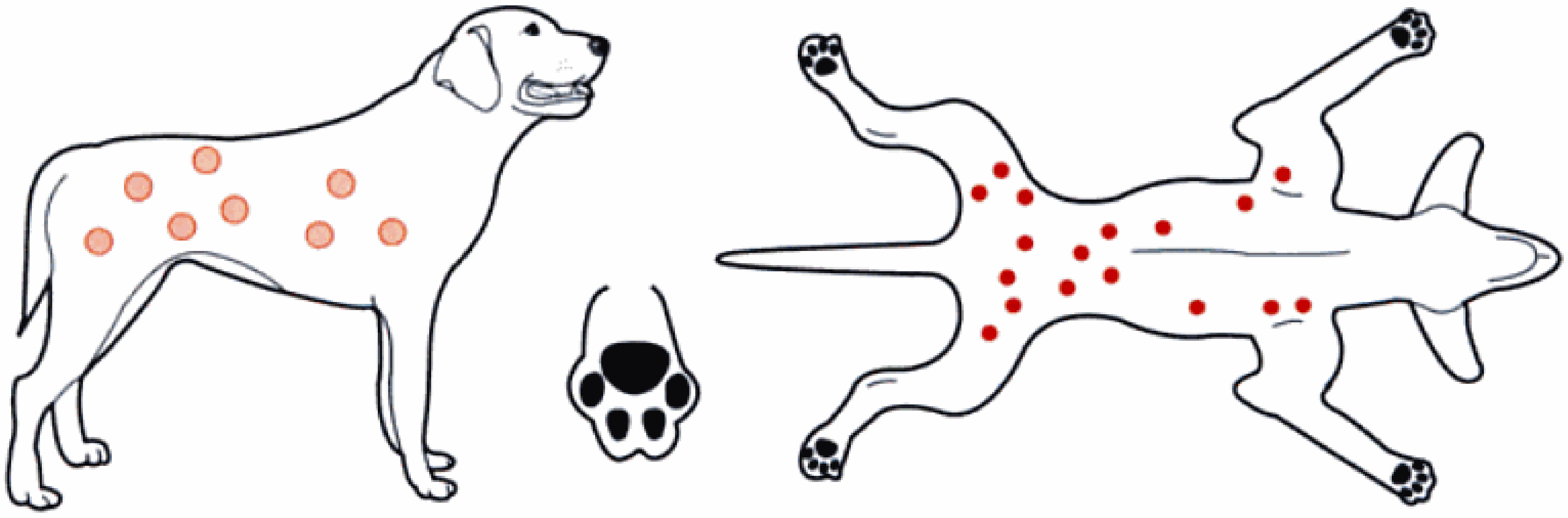
Aetiology: Infection of the skin caused by the bacterium *Staphylococcus intermedius*. Usually secondary to an underlying cause (parasite, allergy, endocrine disorder, immunodeficiency).

Predominant lesions:

Papules, pustules, epidermal collarettes, staphylococcal rings, circular patches of alopecia.

Lesion distribution:

Ventral abdomen, trunk.



Appropriate diagnostic tests:

Direct examination
Cytology
Bacterial culture if doesn't respond to treatment

Malassezia dermatitis

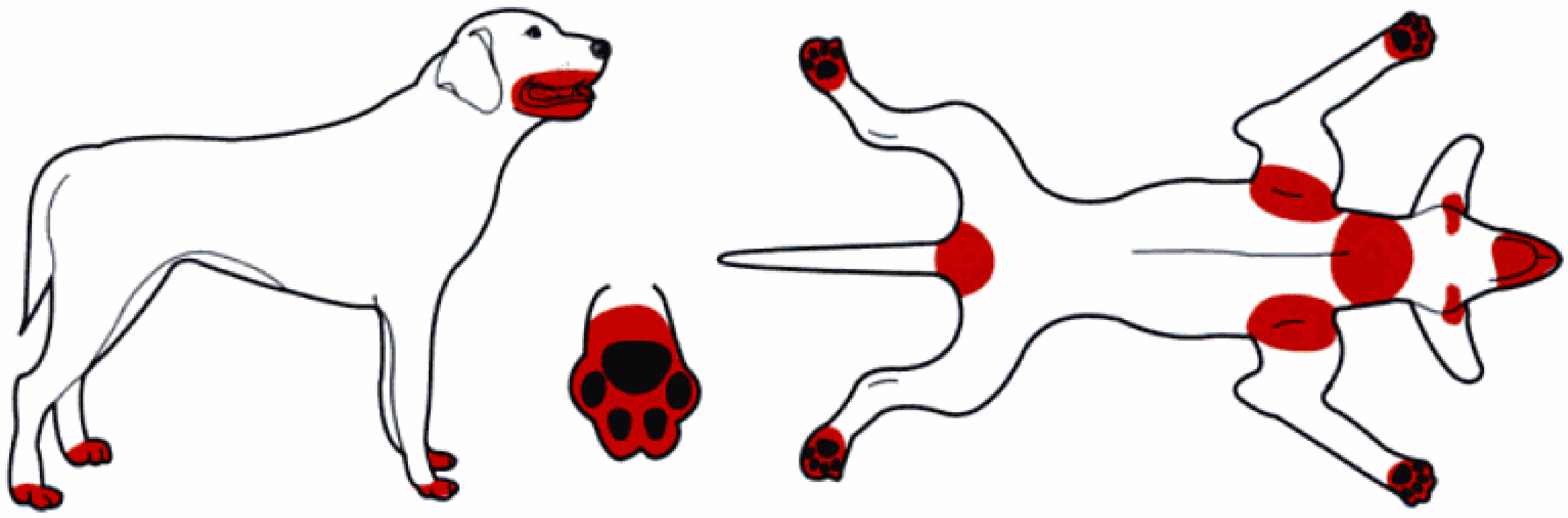
Aetiology: Cutaneous overgrowth of the yeast *Malassezia pachydermatis*. Usually secondary to an underlying cause (parasite, allergy, endocrine disorder, immunodeficiency).

Predominant lesions:

Erythema, yellowish or brownish greasy scale, hyperpigmentation.

Lesion distribution:

Interdigital skin, ventral neck, axillae, muzzle, perineum, ear canals.



Appropriate diagnostic tests:

Cytology

Dermatophytosis

Although dermatophytosis should be considered in the differential diagnosis of pruritus (especially in cats), it most commonly presents as alopecia (see Chapter 5 for further details).

Atopic dermatitis

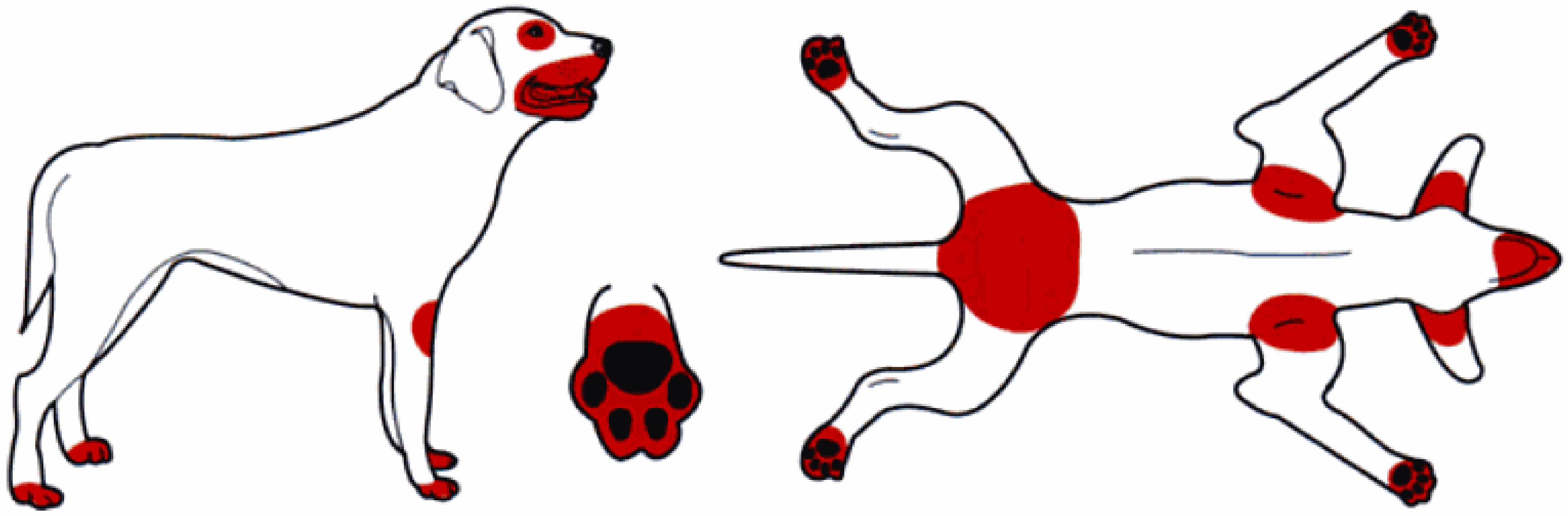
Aetiology: An inherited tendency to produce IgE antibodies against environmental allergens (house dust mites, epidermal antigens, pollens, moulds) resulting in a pruritic skin disease.

Predominant lesions:

Erythema, excoriations, lichenification, hyperpigmentation. Often complicated by secondary staphylococcal and Malassezia infections.

Lesion distribution:

Periocular, face, muzzle, medial aspects of the pinnae, axillae, ventral abdomen, cranial elbows, interdigital skin.



Appropriate diagnostic tests:

Intradermal allergy test
Measurement of allergen-specific IgE by ELISA

Food allergy or intolerance

Aetiology: Adverse reaction to a dietary antigen caused either by immunological or non-immunological mechanisms.

Predominant lesions:

The same as for atopic dermatitis.

Lesion distribution:

The same as for atopic dermatitis.

Appropriate diagnostic tests:

Restricted diet trial

Contact dermatitis

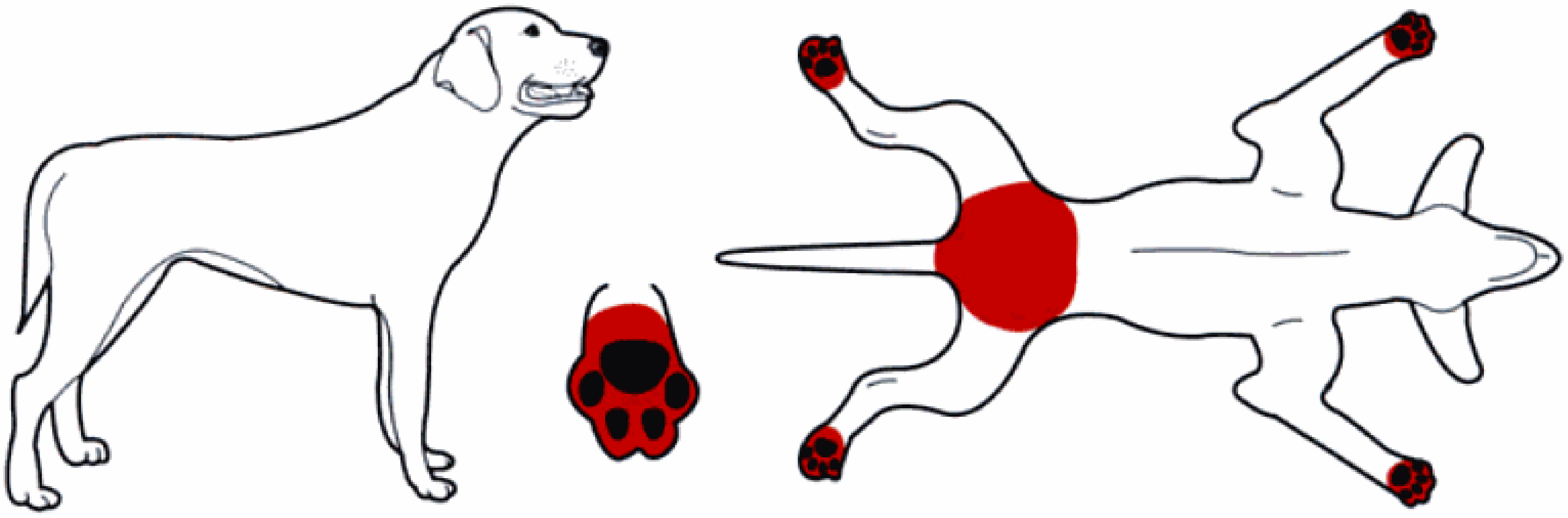
Aetiology: Irritant or allergic reaction to something that contacts the skin.

Predominant lesions:

Erythematous macules, papules, lichenification, hyperpigmentation, erosions.

Lesion distribution:

Typically non-haired areas of the body or other contact points if materials applied Ventral aspects of the feet, scrotum, ventral abdomen.



Appropriate diagnostic tests:

Environmental restriction
Patch testing

Epitheliotropic lymphoma

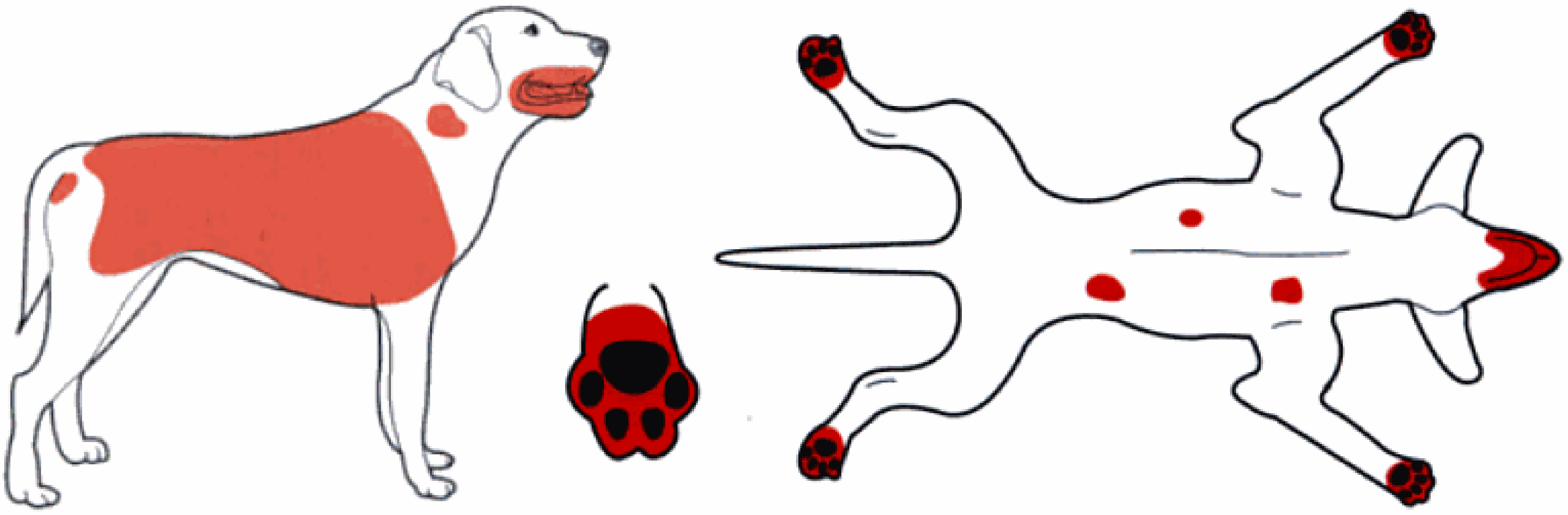
Aetiology: Infiltration of the surface and follicular epithelium by neoplastic lymphocytes.

Predominant lesions:

Scaling, erythroderma, alopecia, exfoliation, hypopigmentation, plaques, nodules, ulcers.

Lesion distribution:

May be focal, generalised or have a mucocutaneous distribution.



Appropriate diagnostic tests:

Cytology
Biopsy

Feline cutaneous reaction patterns

Aetiology: Any of the pruritic diseases in Table 4.1 can lead to any of the following reaction patterns. Flea-bite hypersensitivity is the most common cause, but other ectoparasites, allergies or dermatophytosis can also be involved. Staphylococcal infections and Malassezia overgrowth are much less common in cats than they are in dogs but they can occur as secondary infections in the eosinophilic granuloma complex. Despite an extensive investigation, some cases are idiopathic.

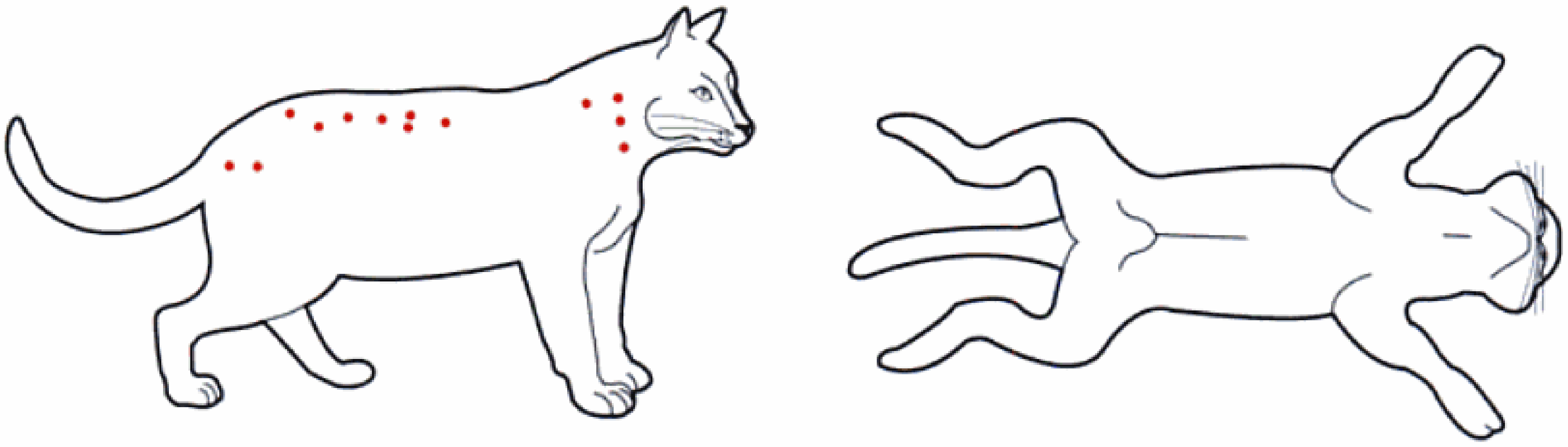
Miliary dermatitis

Predominant lesions:

Crusted papules that may be easier to feel than see.

Lesion distribution:

Usually over the dorsum and around the neck.



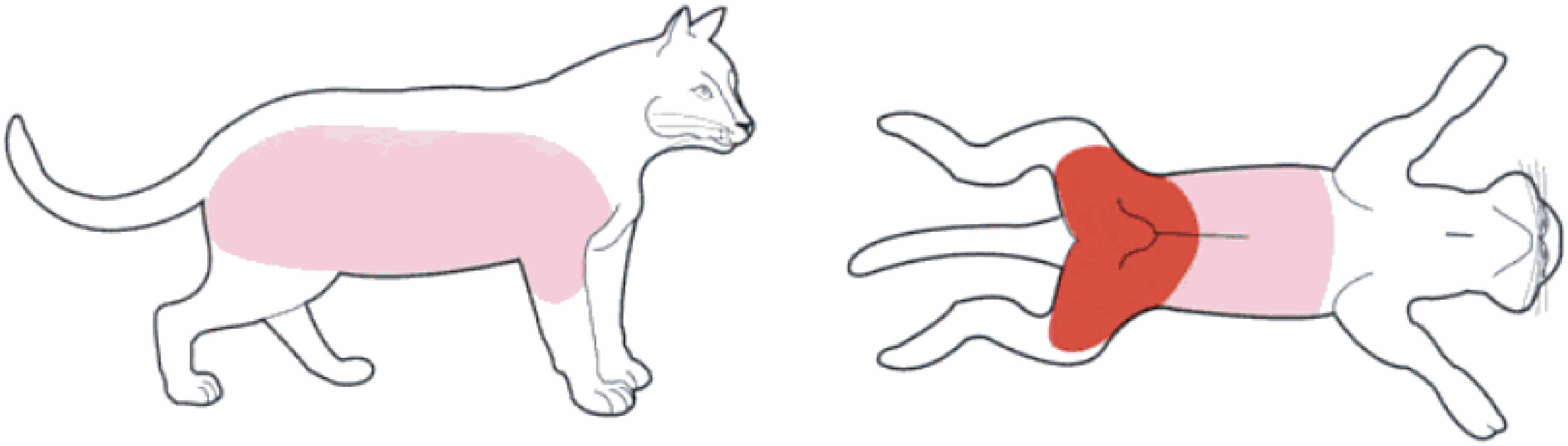
Symmetrical alopecia

Predominant lesions:

Non-inflammatory alopecia caused by licking.

Lesion distribution:

Most commonly ventral abdomen and medial thighs. May extend to ventral chest, lateral chest and abdomen, and lateral thighs.



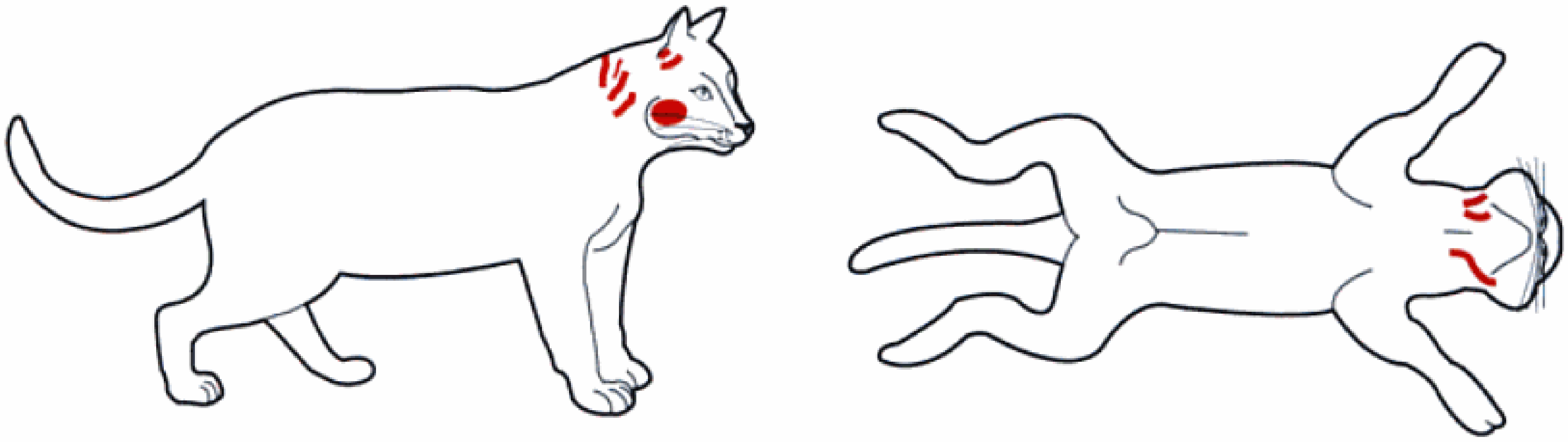
Head and neck pruritus

Predominant lesions:

Excoriations, ulcers.

Lesion distribution:

Around the neck and face.



The eosinophilic granuloma complex

Feline skin is very prone to developing eosinophilic inflammation. Three well-recognised clinical syndromes make up the eosinophilic granuloma complex. These are the:

1. Indolent ulcer
2. Eosinophilic plaque
3. Linear granuloma

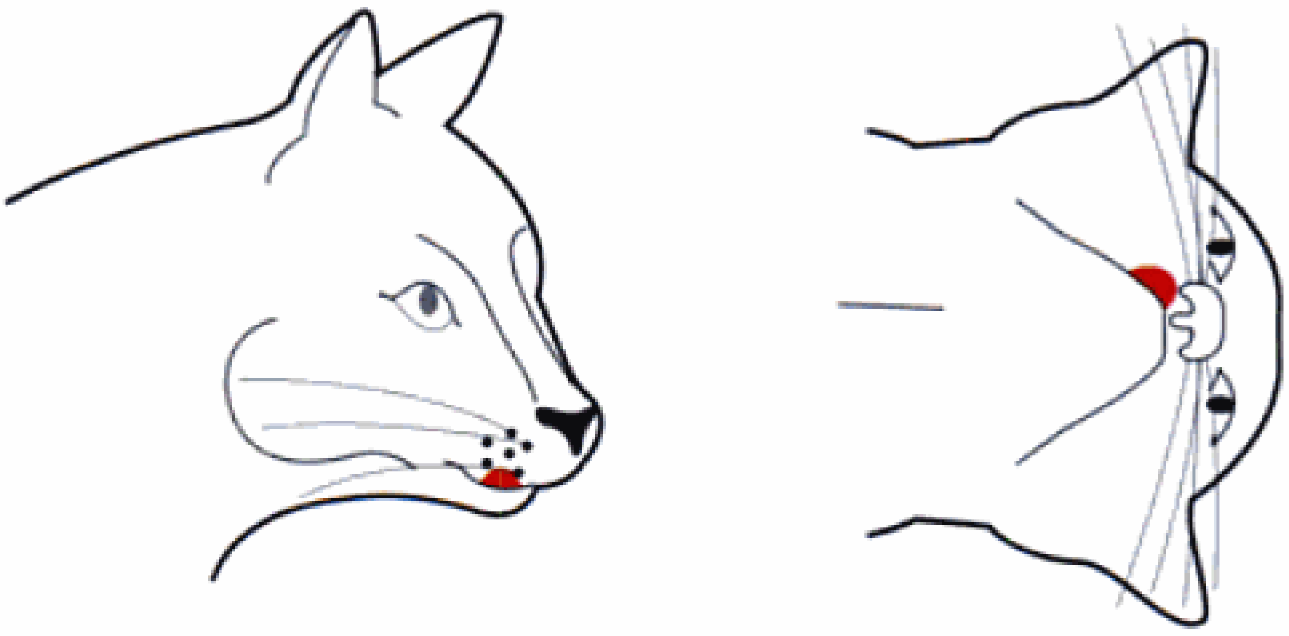
1. Indolent ulcer

Predominant lesions:

Well-circumscribed, red-brown, glistening ulcer with a raised border.

Lesion distribution:

Usually on the upper lip adjacent to the canine tooth.



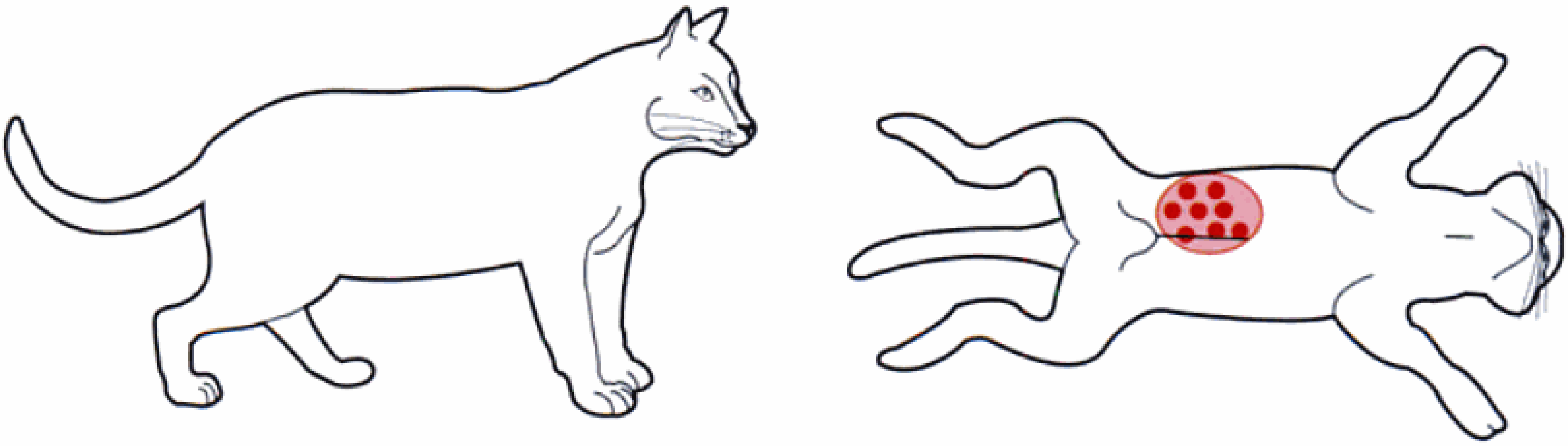
2. Eosinophilic plaque

Predominant lesions:

Highly pruritic, round to oval, erythematous, alopecic, moist, ulcerative plaques.

Lesion distribution:

Ventral abdomen or thorax, medial thighs, lateral thorax.



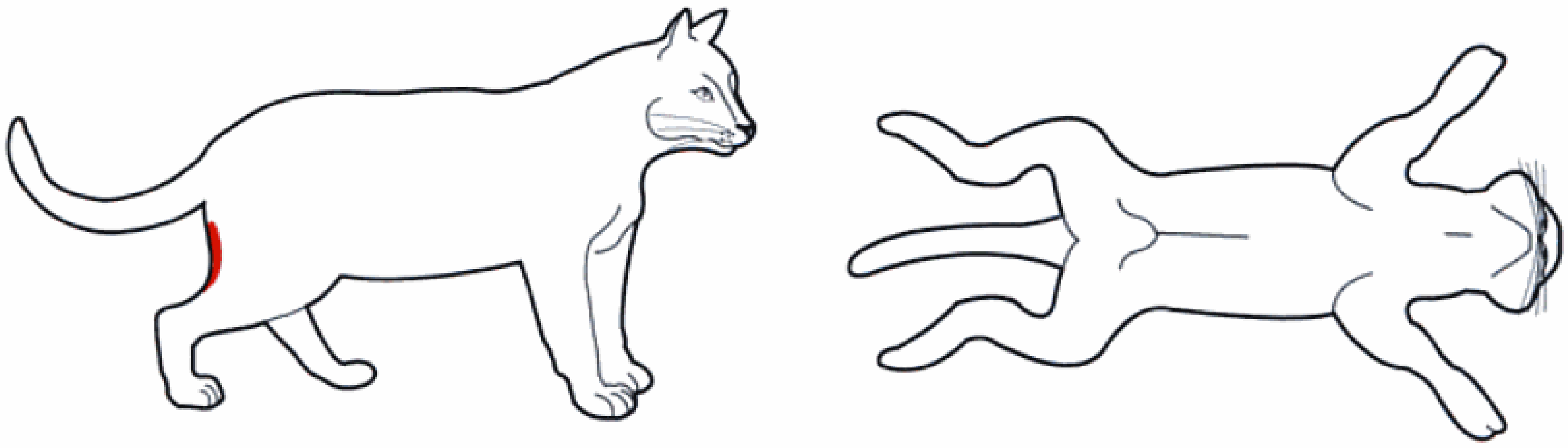
3. Linear granuloma

Predominant lesions:

Linear, cord-like thickening with an erythematous or ulcerated surface.

Lesion distribution:

Caudal aspect of the hindlimb or occasionally in the oral cavity.



Appropriate diagnostic tests for feline reaction patterns:

Cytology
Trichogram
Direct examination for parasites
Coat brushing
Skin scraping
Response to flea control programme
Unstained adhesive tape strip
Wood's lamp examination
Fungal culture
Restricted diet trial
Intradermal allergy test
Biopsy

Hot spots and lick granulomas

Two additional forms of visually distinctive, localised pruritus seen only in dogs warrant further mention. These are:

- Acute moist dermatitis (hot-spots, pyotraumatic dermatitis, acute exudative dermatitis, wet eczema)
- Acral lick dermatitis (lick granuloma)

Both of these conditions have multiple possible aetiologies (Table 4.2).

Acute moist dermatitis

Predominant lesions:

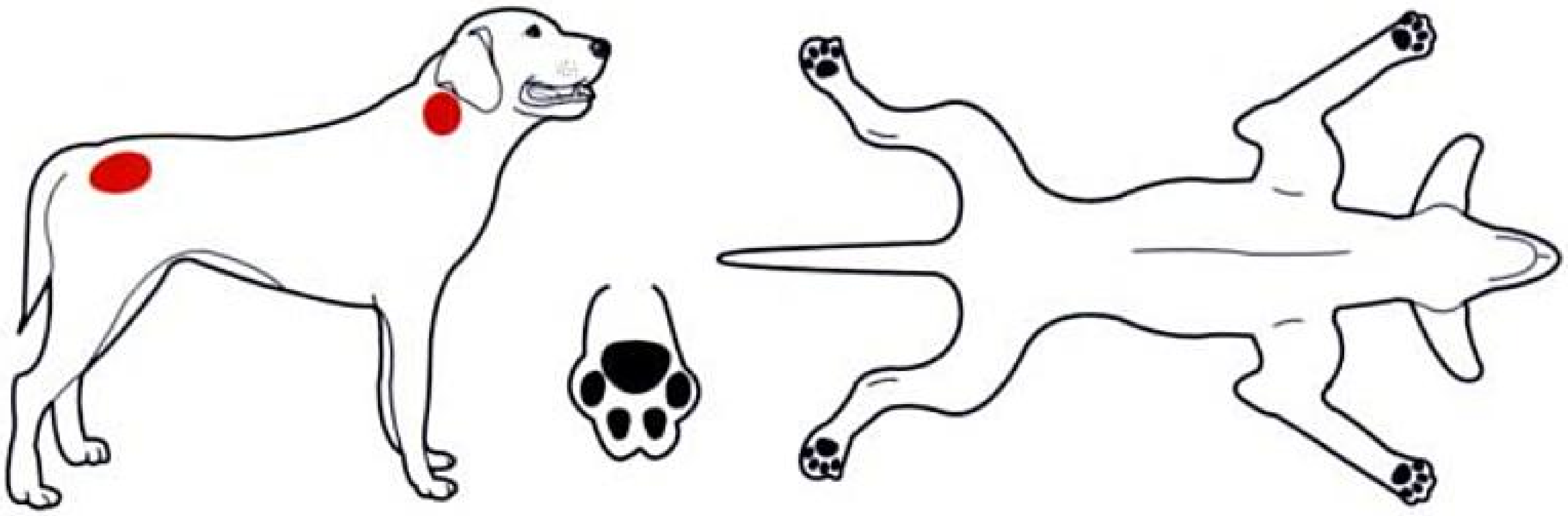
Highly pruritic, well circumscribed, red, exudative erosion, often covered by matted fur. Caused by self-trauma.

Lesion distribution:

Over the rump or on the neck and face.

Table 4.2 Differential diagnoses for acute moist dermatitis and acral lick dermatitis

Presenting condition	Possible underlying causes
Acute moist dermatitis (D)	Flea bite hypersensitivity Anal sacculitis Otitis externa Atopic dermatitis Food intolerance Scabies Staphylococcal pyoderma
Acral lick dermatitis (D)	Staphylococcal pyoderma Demodicosis Fungal infection Trauma Foreign bodies Atopic dermatitis Food intolerance Hypothyroidism Underlying joint disease Nerve dysfunction Anxiety, boredom, separation Obsessive compulsive disorder



Appropriate diagnostic tests:

Cytology
Direct examination for parasites
Coat brushing
Skin scraping
Response to symptomatic treatment and flea control
Response to symptomatic treatment and anal sac expression
Otoscopic examination
Restricted diet trial
Intradermal allergy testing

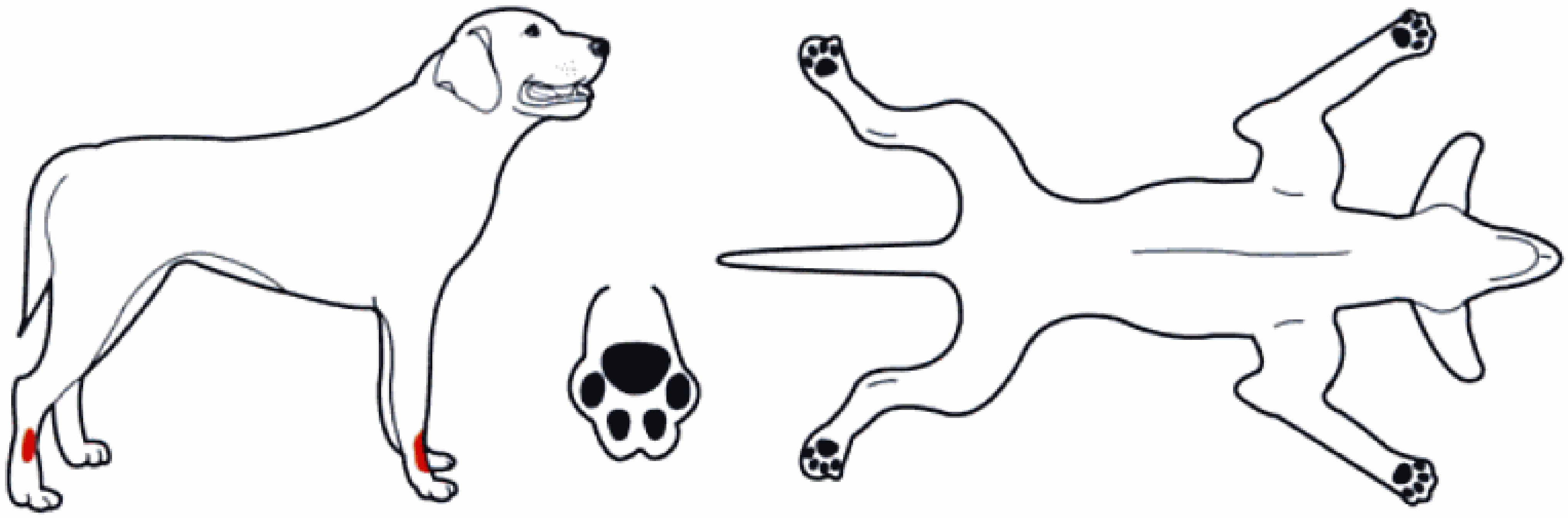
Acral lick dermatitis

Predominant lesions:

Well-circumscribed, alopecic, ulcerated plaque caused by self-trauma.

Lesion distribution:

Distal limbs.



Appropriate diagnostic tests:

Cytology
Skin scraping
Bacterial culture
Fungal culture
Restricted diet trial
Intradermal allergy test
Thyroid function tests
Radiography
Neurological examination
Nerve conduction studies

General diagnostic approach to pruritic skin diseases

In many cases of pruritic skin disease, the history and clinical signs will be suggestive of a particular condition. In these cases, the diagnosis can usually be confirmed by the use of a few appropriate diagnostic tests. However, in other cases, making a diagnosis is not so simple. This usually occurs when:

- The animal has more than one disease at the same time. This leads to over-lapping of the clinical signs and lesion distribution patterns. A typical example would be a dog with atopic dermatitis and secondary staphylococcal and Malassezia infections.
- The animal has a clinical presentation that can be caused by multiple diseases. An example would be the feline cutaneous reaction patterns.
- The animal has a disease that can closely resemble another one. An example would be the over-lapping lesion distribution patterns of atopic dermatitis, scabies and Malassezia dermatitis.

In these more difficult cases, it is important to follow a logical diagnostic sequence to rule out the various possibilities. Figure 4.1 shows a diagnostic algorithm that can be used to investigate such cases. It is important to remember that this algorithm is only a guide and the clinician must use the information obtained from the history and physical examination to prioritise the initial differential diagnosis list. This may allow some of the steps to be by-passed. For example, if a dog presented solely for foot chewing, it would not be necessary

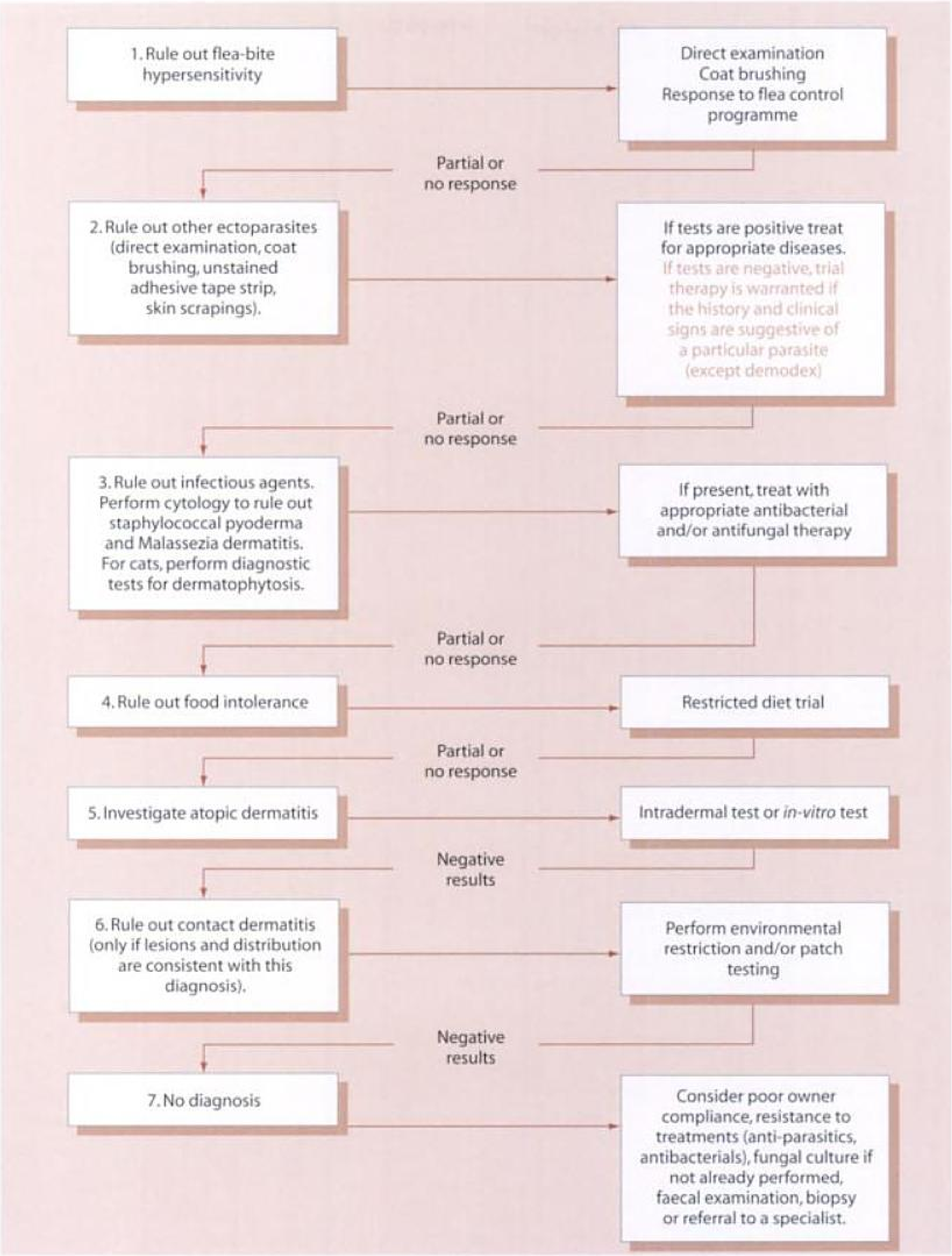

1. Rule out flea-bite hypersensitivity
Direct examination Coat brushing Response to flea control programme
Partial or no response
2. Rule out other ectoparasites (direct examination, coat brushing, unstained adhesive tape strip, skin scrapings).
If tests are positive treat for appropriate diseases. If tests are negative, trial therapy is warranted if the history and clinical signs are suggestive of a particular parasite (except demodex)
Partial or no response
3. Rule out infectious agents. Perform cytology to rule out staphylococcal pyoderma and Malassezia dermatitis. For cats, perform diagnostic tests for dermatophytosis.
If present, treat with appropriate antibacterial and/or antifungal therapy
Partial or no response
4. Rule out food intolerance
Restricted diet trial
Partial or no response
5. Investigate atopic dermatitis
Intradermal test or in-vitro test
Negative results
6. Rule out contact dermatitis (only if lesions and distribution are consistent with this diagnosis).
Perform environmental restriction and/or patch testing
Negative results
7. No diagnosis
Consider poor owner compliance, resistance to treatments (anti-parasitics, antibacterials), fungal culture if not already performed, faecal examination, biopsy or referral to a specialist.

Figure 4.1 General diagnostic approach to pruritus.

to rule out flea bite hypersensitivity and step 1 could be avoided. Also, if the diagnostic tests have yielded positive results, it is possible to combine steps 1 to 3. This should not be done if the clinician has not made a positive diagnosis and is using 'response to treatment' as a diagnostic test.

5

Alopecia

Differential diagnosis

Alopecia is a common presenting sign in canine and feline dermatology. In many cases, the hair loss is caused by the animal scratching, licking or chewing. Hence, if the animal is

Table 5.1 Differential diagnoses for alopecia

Pattern of alopecia	Specific disease
Localised alopecia (a single patch of alopecia)	**Demodicosis (D and C)**
	Dermatophytosis (D and C)
	Scars (D and C)
	Medication application site (D and C)
	Steroid injection site (D and C)
	Alopecia areata (D)
	Post-clipping alopecia (D)
Multifocal alopecia (appears as patchy, circular areas of alopecia. Coat looks 'moth-eaten')	**Demodicosis (D and C)**
	Staphylococcal pyoderma (D)
	Dermatophytosis (D and C)
	Sebaceous adenitis (D)
	Colour dilution alopecia (D)
	Dermatomyositis (D)
Symmetrical or diffuse alopecia	**Demodicosis (D and C)**
	Dermatophytosis (D and C)
	Hypothyroidism (D)
	Hyperadrenocorticism (D and C)
	Gonadal sex hormone alopecia (D)
	GH/adrenal sex hormone alopecia (D)
	Pituitary dwarfism (D)
	Cyclic flank alopecia (D)
	Follicular dysplasia (D)
	Colour dilution alopecia (D)
	Black hair follicular dysplasia (D)
	Pattern baldness (D)
	Congenital alopecia (D and C)
	Telogen/anagen defluxion (D)
	Sebaceous adenitis (D)
	Epitheliotropic lymphoma (D and C)
	Post-clipping alopecia (D)
	Paraneoplastic alopecia (C)
	Feline symmetrical alopecia (C)

Those in bold would be most common in general practice. D = those diseases seen in dogs, C = those diseases seen in cats.

pruritic, this should be investigated first (see Chapter 1). This chapter is concerned with non-pruritic, spontaneous hair loss.

When investigating the possible causes of alopecia, it is useful to divide the differential diagnoses into categories based on their presenting pattern. Table 5.1 does this by showing the major differential diagnoses for localised alopecia, multi-focal alopecia and symmetrical alopecia.

Clinical features of alopecic skin diseases

Demodicosis (Demodectic mange)

Aetiology: Excessive proliferation of demodex mites in the hair follicles or on the skin surface. Demodicosis in juvenile dogs is thought to be due to a defect in the skin immune system. Adult-onset demodicosis (over 4 years of age) may be secondary to hyperadrenocorticism, hypothyroidism, other internal diseases, malignancies, excessive use of glucocorticoids or idiopathic.

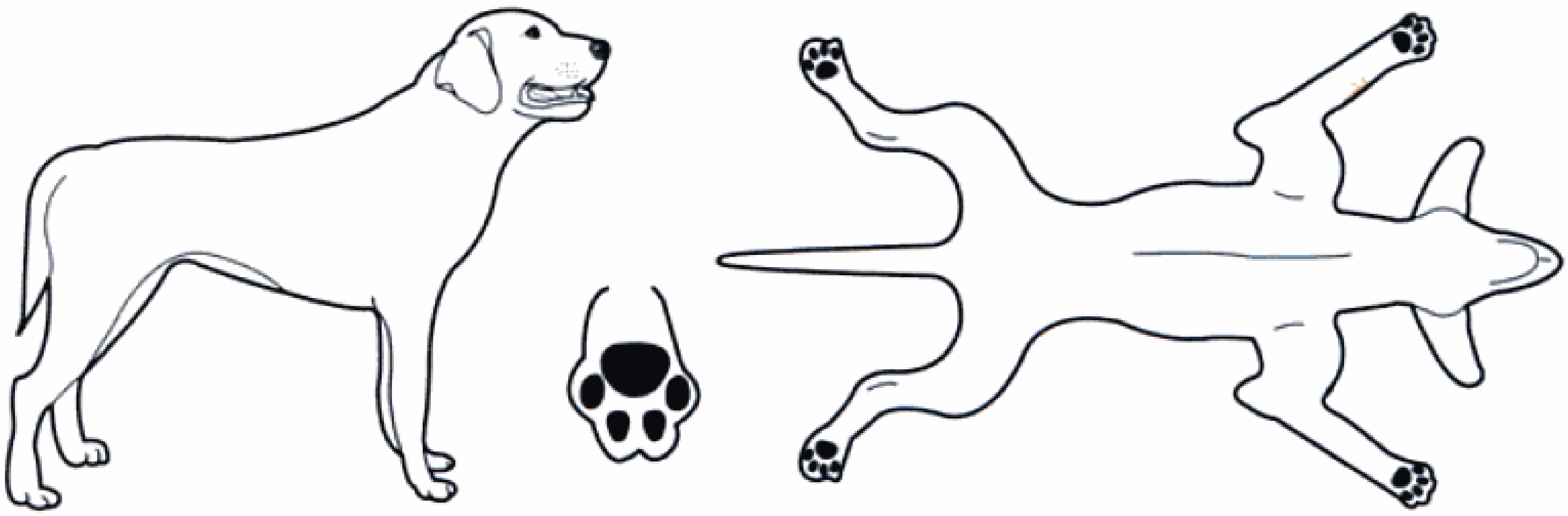
Predominant lesions:

Patches of alopecia, comedones, follicular casts, erythema, scale, slate-grey hyperpigmentation. Papules, pustules, furunculosis and ulcers if secondary infection present.

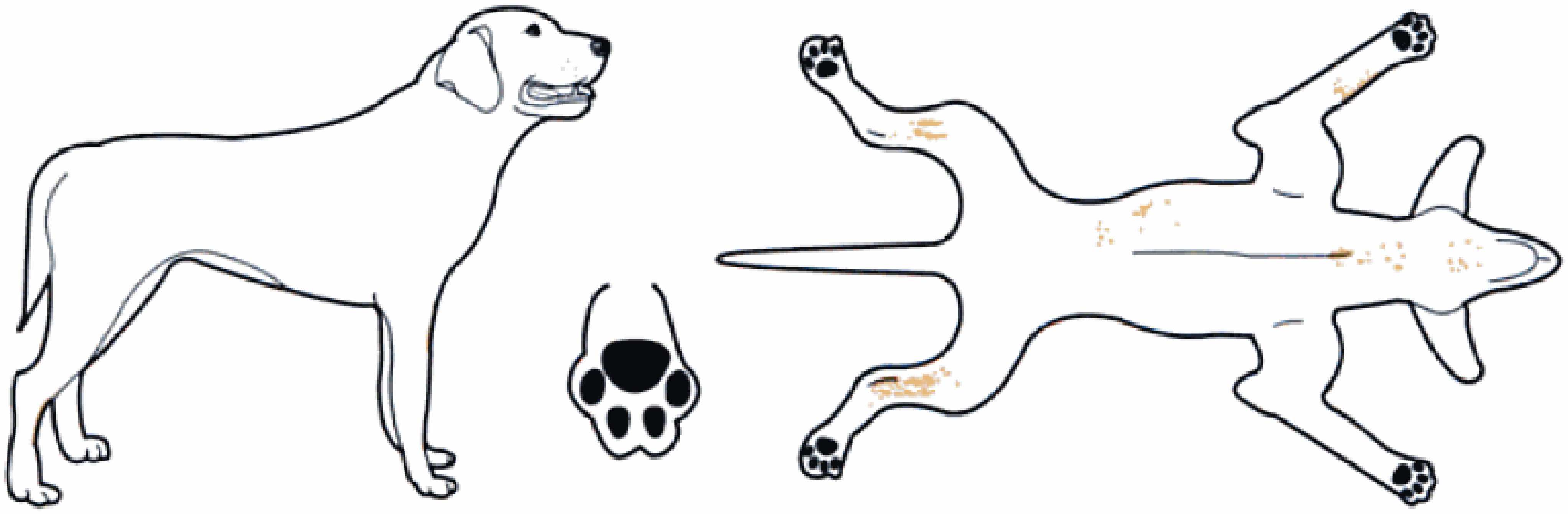
Lesion distribution:

Three main forms of demodicosis are seen, but there is overlap between the presentations:

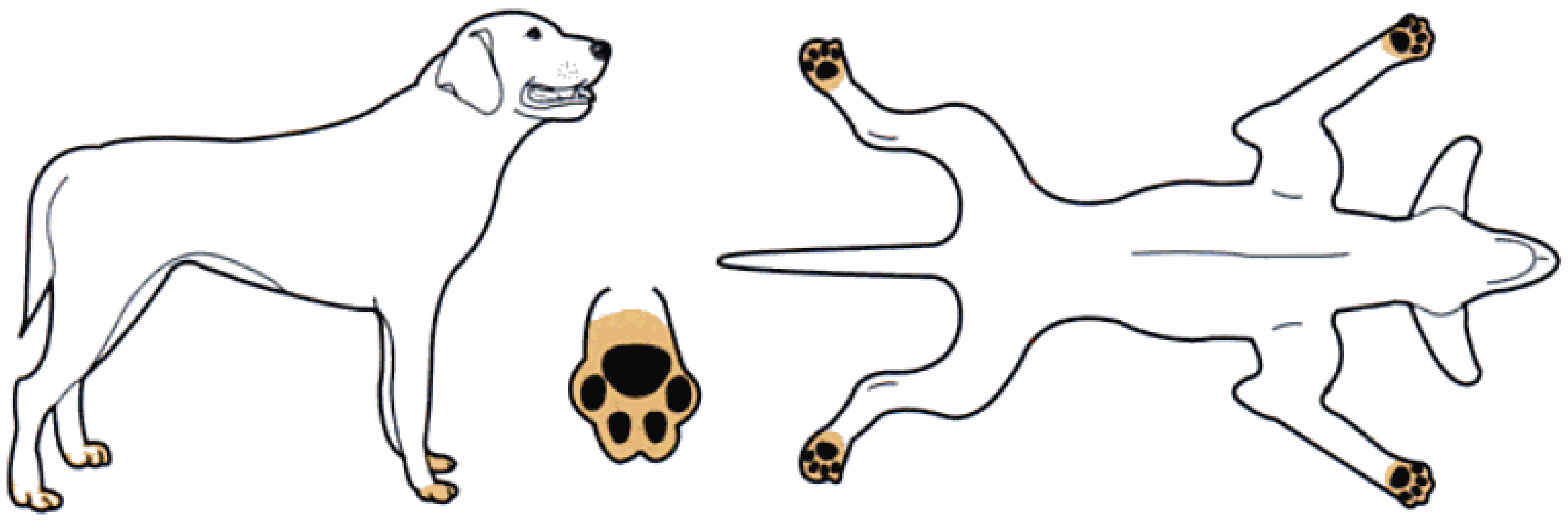
1. Localised demodicosis – Few focal patches of alopecia on the face and legs.



2. Generalised demodicosis – Multifocal or diffuse lesions over the face, legs and trunk.



3. Demodectic pododermatitis – Lesions on the feet.



Appropriate diagnostic tests:

Skin scraping
Trichogram
Biopsy

Dermatophytosis

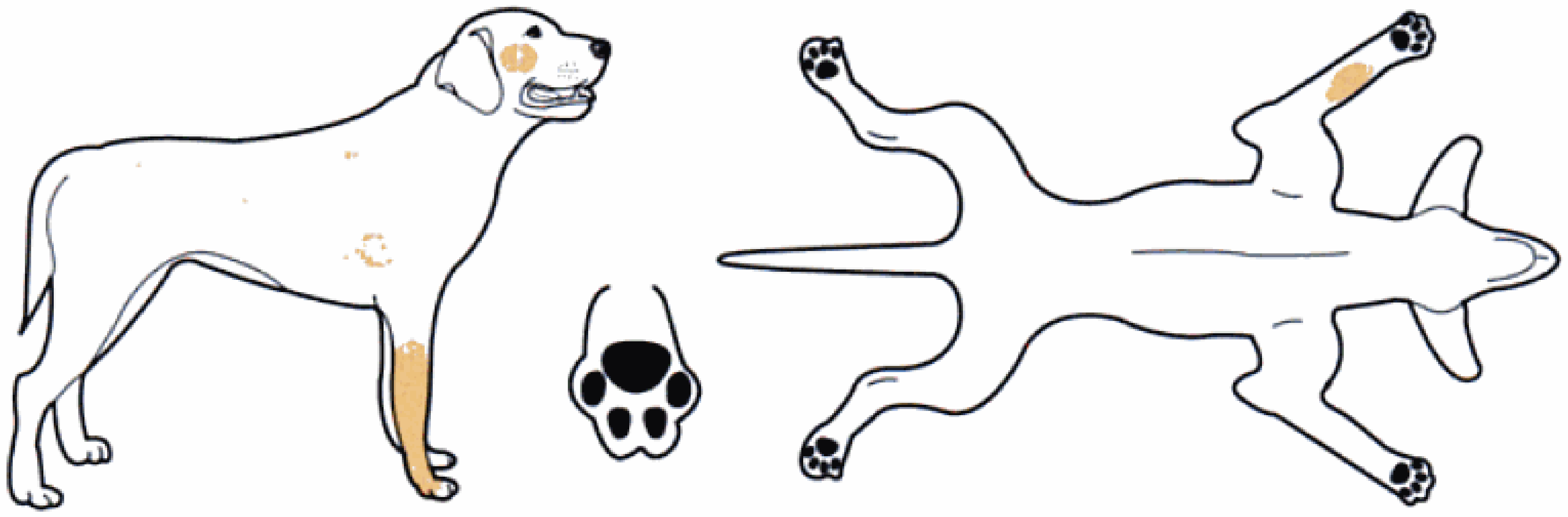
Aetiology: Infection with Microsporum or Trichophyton fungi.

Predominant lesions:

Patches of alopecia and scaling, with or without erythema.

Lesion distribution:

May be focal, multifocal or diffuse.



Appropriate diagnostic tests:

Wood's lamp examination
Trichogram
Fungal culture
Biopsy

Superficial pyoderma (superficial folliculitis, staphylococcal pyoderma)

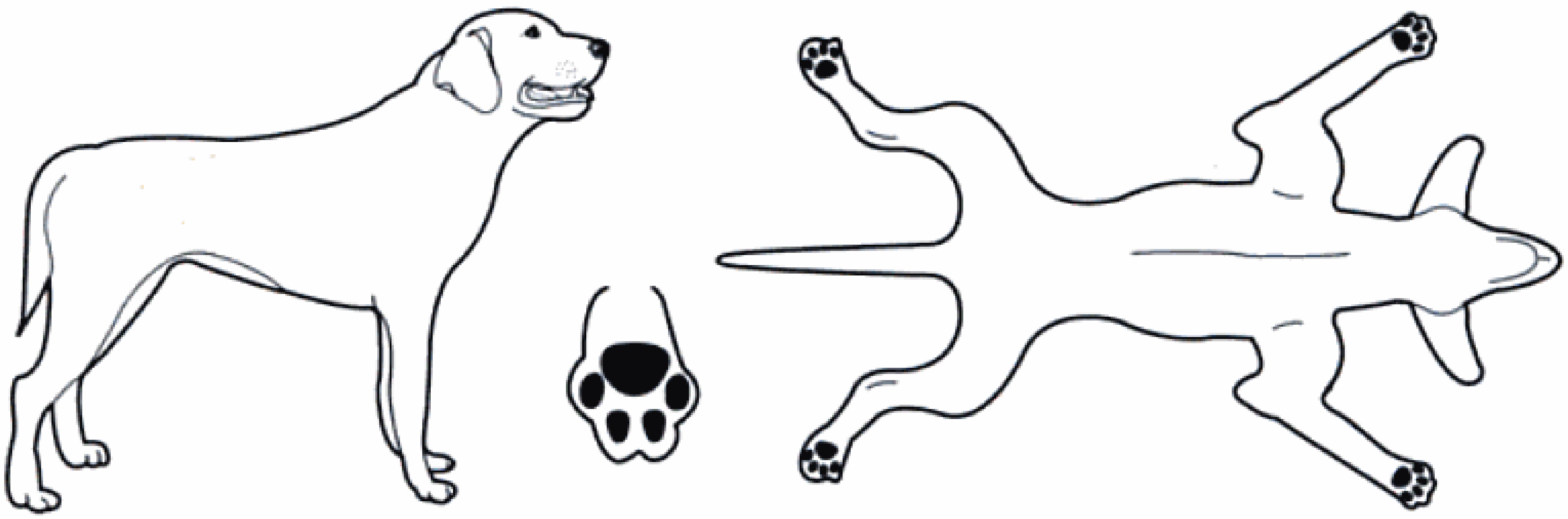
Aetiology: see Chapter 4.

Predominant lesions:

Staphylococcus intermedius usually causes a pruritic papulo-pustular eruption. However, in some dogs, especially short-coated breeds, the predominant lesions can be non-inflammatory, circular patches of alopecia with peripheral scaling (epidermal collarettes).

Lesion distribution:

Multi-focal over the trunk.



Appropriate diagnostic tests:

Direct examination
Cytology
Bacterial culture if doesn't respond to treatment

Dermatomyositis

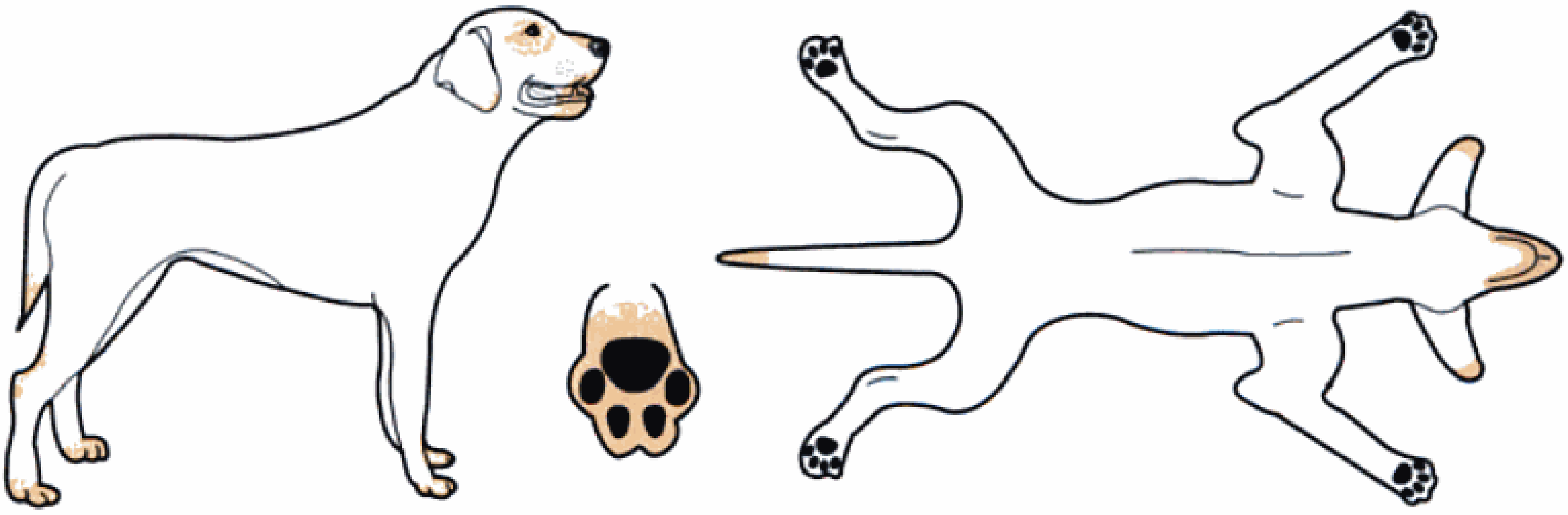
Aetiology: A rare disorder usually seen in Collies and Shetland Sheepdogs but rarely reported in other breeds. May be associated with genetic, immune-mediated or viral factors.

Predominant lesions:

Patches of alopecia and scaling that may progress to crusting and ulceration. Dogs may also develop dysfunction of facial, pharyngeal, oesophageal and leg muscles.

Lesion distribution:

Face, ear tips, distal limbs, end of tail.



Appropriate diagnostic tests:

Biopsy

Alopecia areata

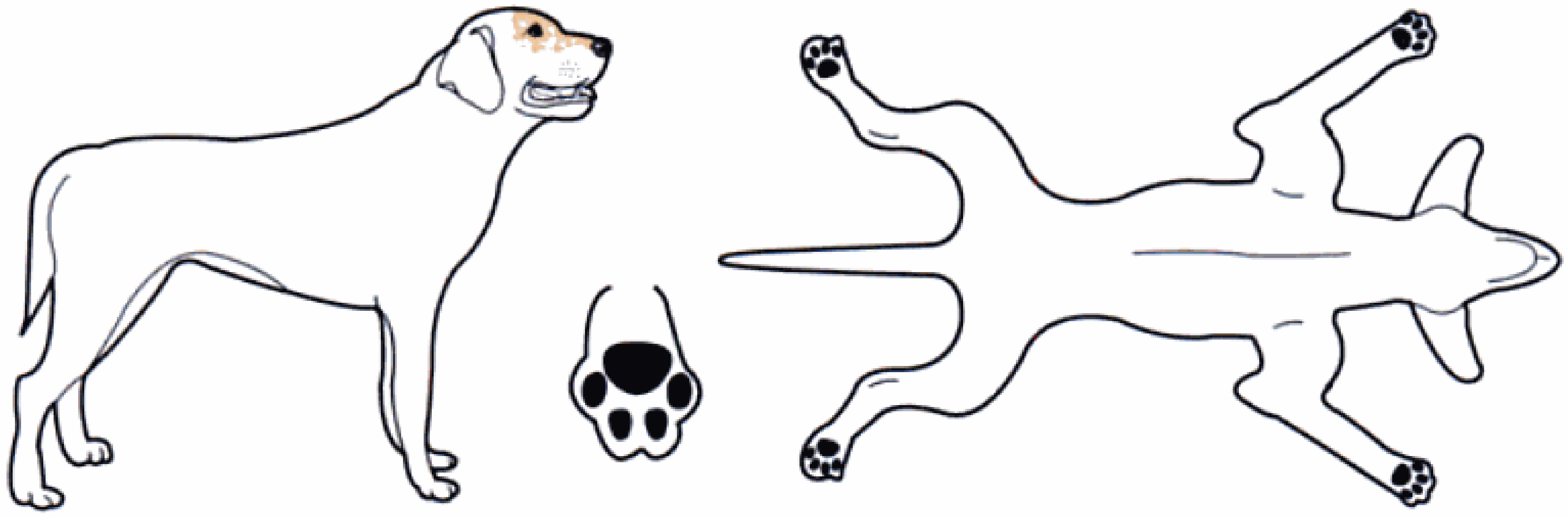
Aetiology: Rare immune-mediated disease in which autoantibodies target the hair follicle.

Predominant lesions:

Focal or multi-focal patches of non-inflammatory alopecia which may regrow white hair.

Lesion distribution:

Usually on the head, neck or trunk and rarely generalised.



Appropriate diagnostic tests:

Trichogram
Biopsy

Post-clipping alopecia

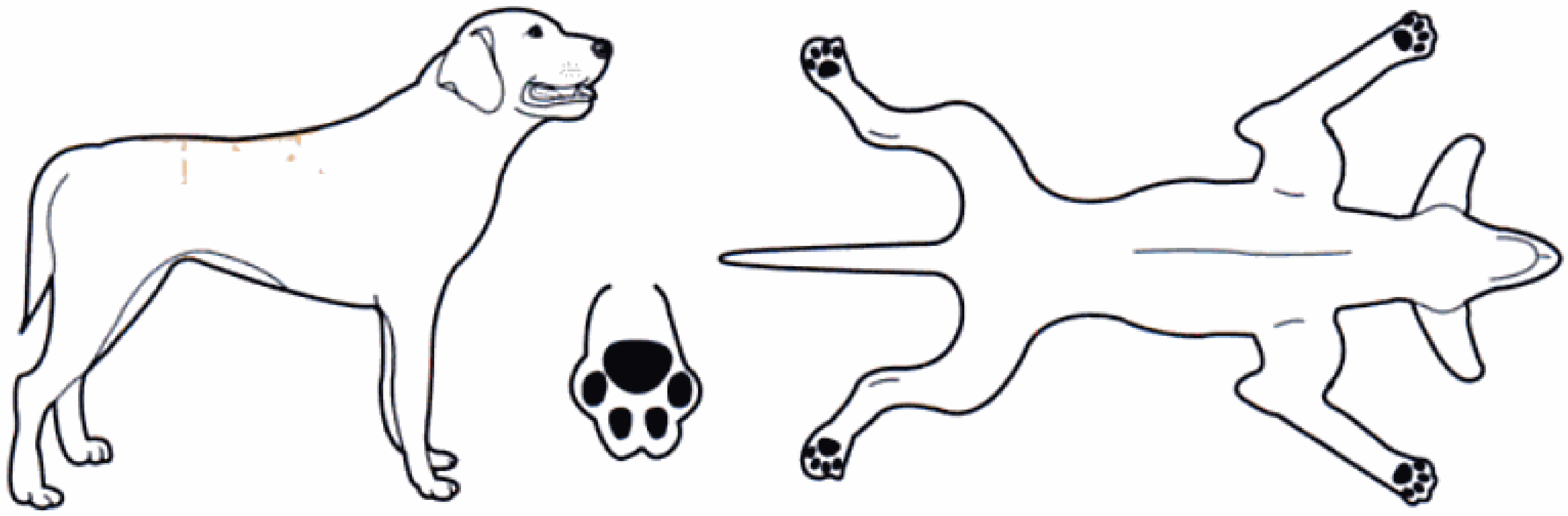
Aetiology: Failure of hair to regrow after clipping for surgery, blood samples, etc. May be due to previously undetected endocrine disorder or synchronisation with the dog's moulting cycle.

Predominant lesions:

Non-inflammatory alopecia.

Lesion distribution:

At the sites of clipping.



Appropriate diagnostic tests:

Trichogram
Endocrine function tests
Biopsy

Medication application or steroid injection site

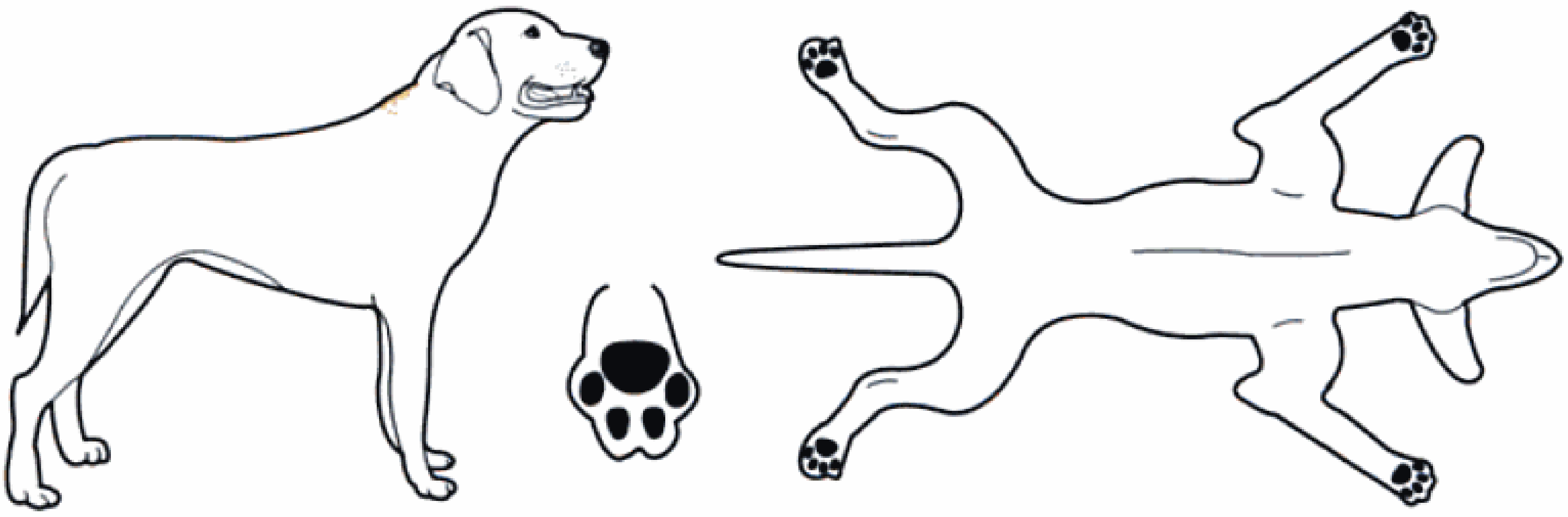
Aetiology: Reaction to spot-on application of various insecticides or subcutaneous injection of depot glucocorticoids.

Predominant lesions:

Focal patch of alopecia.

Lesion distribution:

Site of application or injection.



Appropriate diagnostic tests:

Biopsy

Hypothyroidism

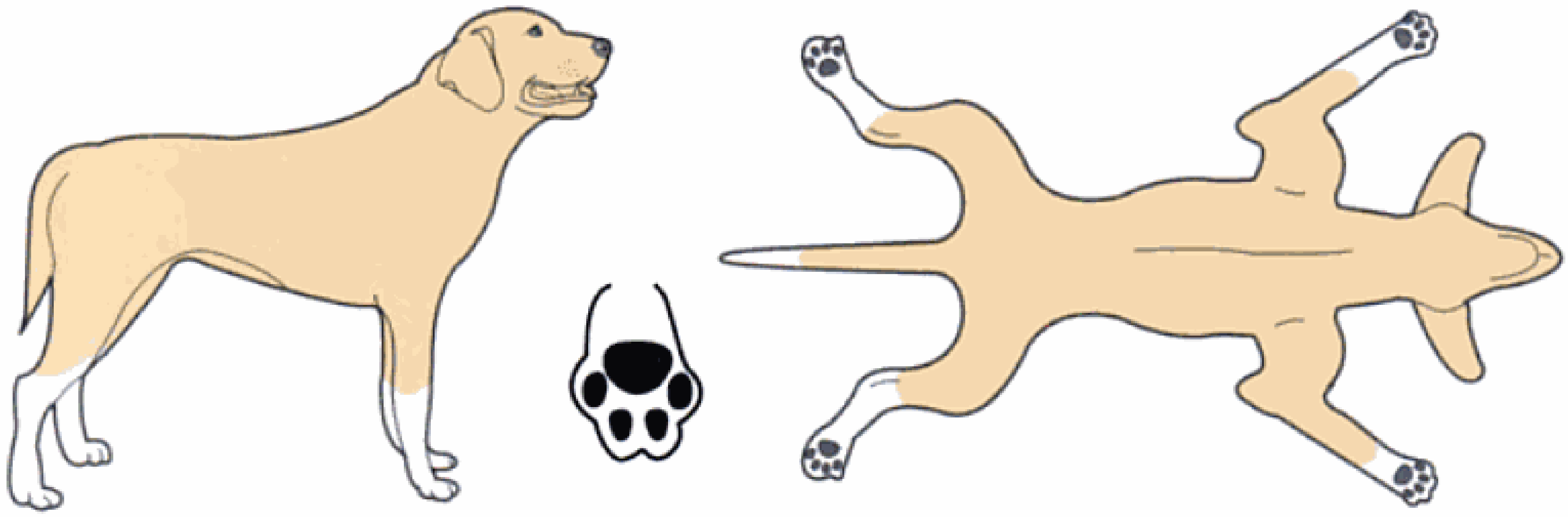
Aetiology: Lymphocytic infiltration or idiopathic atrophy of the thyroid gland leading to inadequate production of thyroid hormones.

Predominant lesions:

Cutaneous – Alopecia (NOT always present), dull dry hair coat, fading coat colour, seborrhoea, comedones, thick skin (myxoedema), cool skin, hyperpigmentation, secondary infections, easy bruising, otitis, occasionally hypertrichosis.
Systemic signs – Lethargy, weight gain, heat-seeking, bradycardia, corneal lipidosis, mild anaemia, neuro-muscular abnormalities, infertility.

Lesion distribution:

Symmetrical and generalised – bridge of nose, face, ears, trunk, ventrum, perineum, tail.



Appropriate diagnostic tests:

Trichogram
Haematology and biochemistry
Thyroid function tests
Biopsy

Hyperadrenocorticism

Aetiology: Excessive production of cortisol from the adrenal glands due to either an ACTH secreting pituitary tumour (85–90% of cases) or functional adrenal tumour (10–15% of cases).

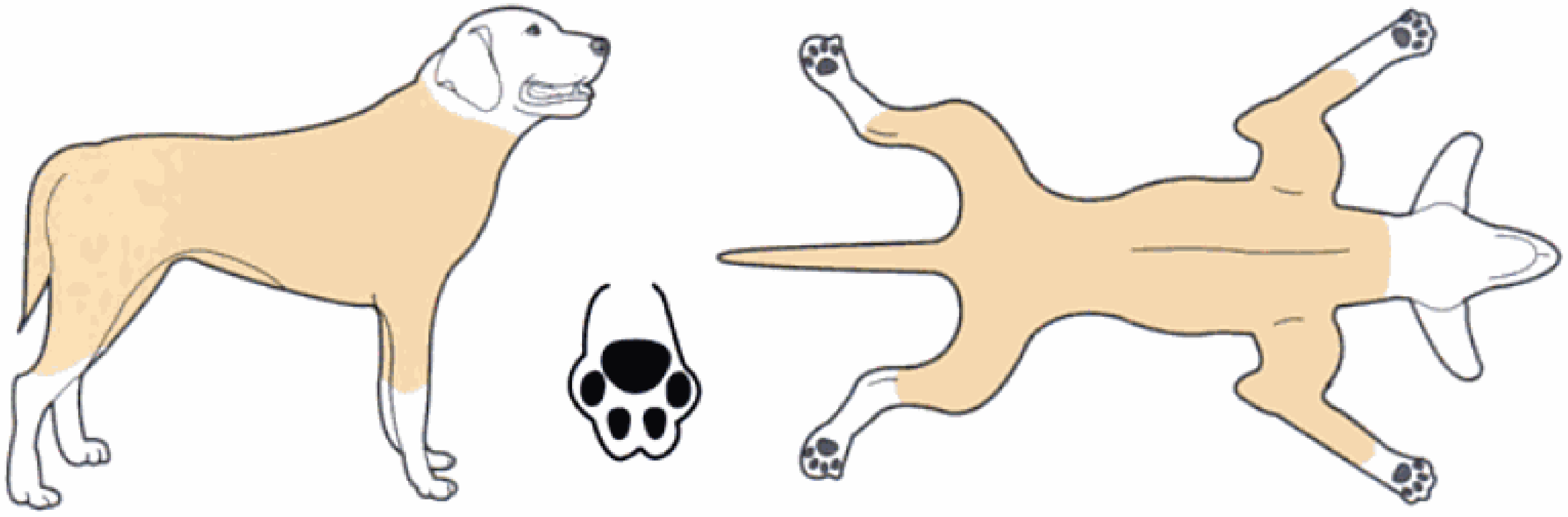
Predominant lesions:

Cutaneous – Alopecia, thin wispy coat, fading coat colour, thin skin, hypotonic skin (tends to wrinkle and mimic dehydration), seborrhoea, comedones, hyperpigmentation, secondary pyoderma, poor wound healing, easy bruising, calcinosis cutis, secondary demodicosis.

Systemic signs – Polydipsia, polyuria, polyphagia, excessive panting, lethargy, muscle atrophy, abdominal distension, neurological signs.

Lesion distribution:

Usually symmetrical, sparing the head and distal limbs. Occasionally, the latter sites may be affected.



Appropriate diagnostic tests:

Trichogram
Haematology and biochemistry
Adrenal function tests
Biopsy
Abdominal ultrasonography
CT and MRI

Gonadal sex hormone alopecia

Aetiology: May be associated with excessive hormone production:

- Testicular neoplasia – Oestrogen-secreting Sertoli's cell tumour or testosterone secreting interstitial cell tumours
- Ovarian neoplasia – oestrogen secreting tumours or cysts

Or presumed lack of hormones or hormone receptors at the hair follicle level:

- Oestrogen-responsive alopecia
- Testosterone-responsive alopecia

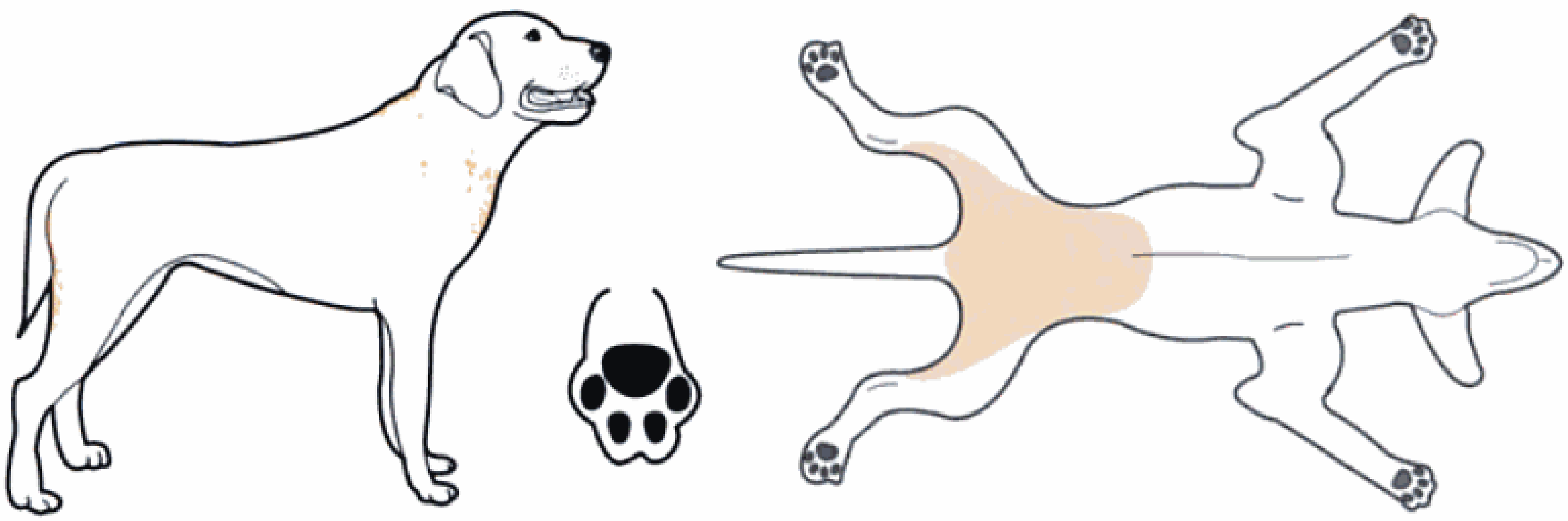
Predominant lesions:

Cutaneous – Alopecia, seborrhoea, comedones, hyperpigmentation, macular melanosis (hyperpigmented macules) of the anus, perineum, scrotum and ventral tail, linear preputial dermatosis (specific for Sertoli's cell tumour), tail gland hyperplasia, circumanal gland hyperplasia.

Systemic signs – May see behaviour changes, nymphomania, feminisation, gynaecomastia, vulval enlargement, pendulous prepuce depending on the hormones involved.

Lesion distribution:

Ventral abdomen, caudal thighs, perineum, neck but may generalise.



Appropriate diagnostic tests:

Trichogram
Sex hormone assays
Biopsy

Growth hormone/adrenal sex hormone alopecia (Castration responsive dermatosis, Alopecia X)

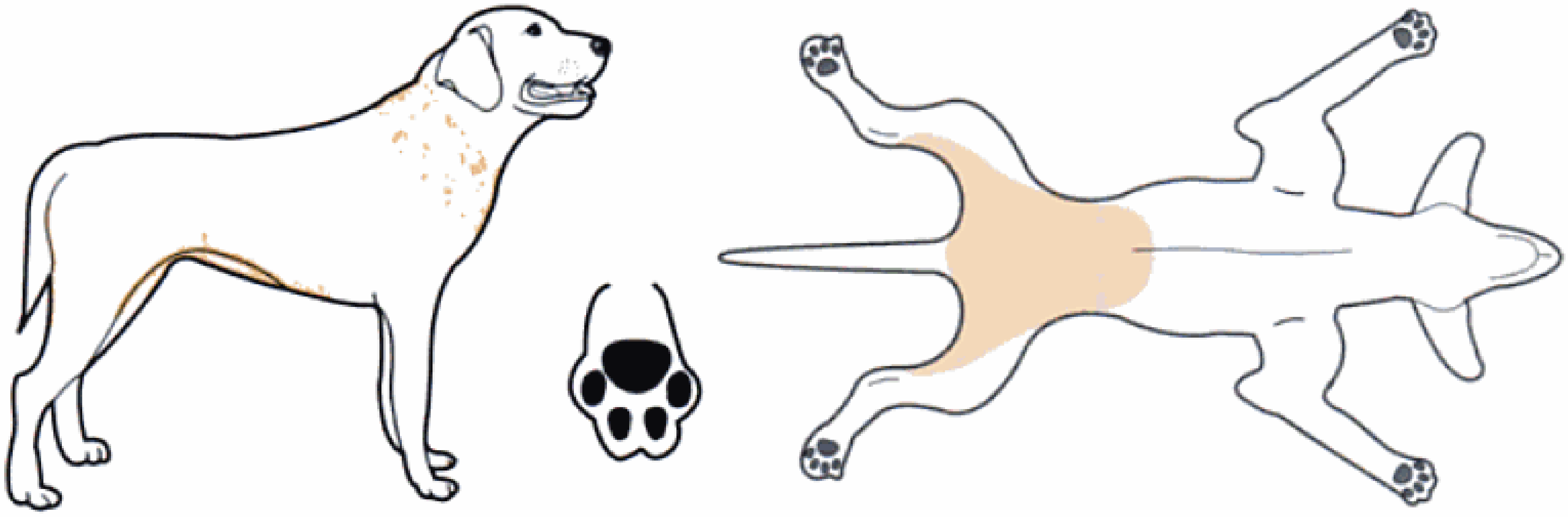
Aetiology: Precise aetiology is unknown, but may involve aberrant production of sex hormones from the adrenal cortex. Most common in the Pomeranian, Chow Chow, Keeshond and Samoyed but can be seen in other breeds.

Predominant lesions:

Alopecia, dull coat, mild scaling, hyperpigmentation.

Lesion distribution:

Ventral abdomen, caudal thighs, tailhead, neck extending to lateral chest and abdomen.



Appropriate diagnostic tests:

Trichogram
Sex hormone assays pre- and post-ACTH
Biopsy

Pituitary dwarfism (hypopituitarism)

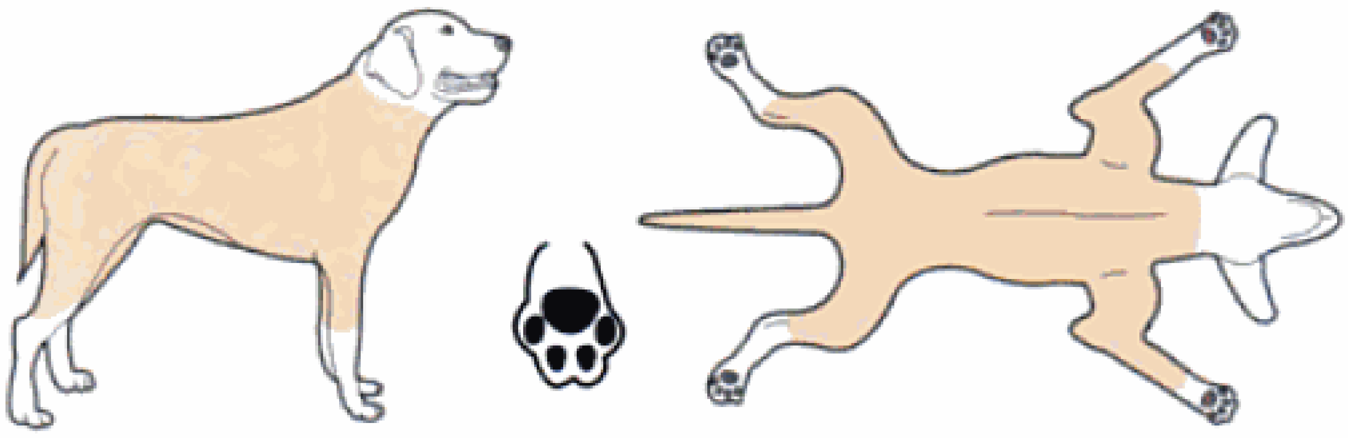
Aetiology: A congenital abnormality seen most commonly in German shepherd dogs caused by an autosomal recessive trait leading to abnormal development of the pituitary gland. Instead of a glandular structure, the pituitary is replaced by a cystic swelling (Cystic Rathke's cleft).

Predominant lesions:

Retention of puppy coat followed by alopecia, mild scaling, and hyperpigmentation
Also proportionate dwarfism, shrill bark, retained deciduous dentition.

Lesion distribution:

Trunk, but sparing head and extremities.



Appropriate diagnostic tests:

Measurement of insulin-like growth factor

Cyclic flank alopecia (Seasonal flank alopecia)

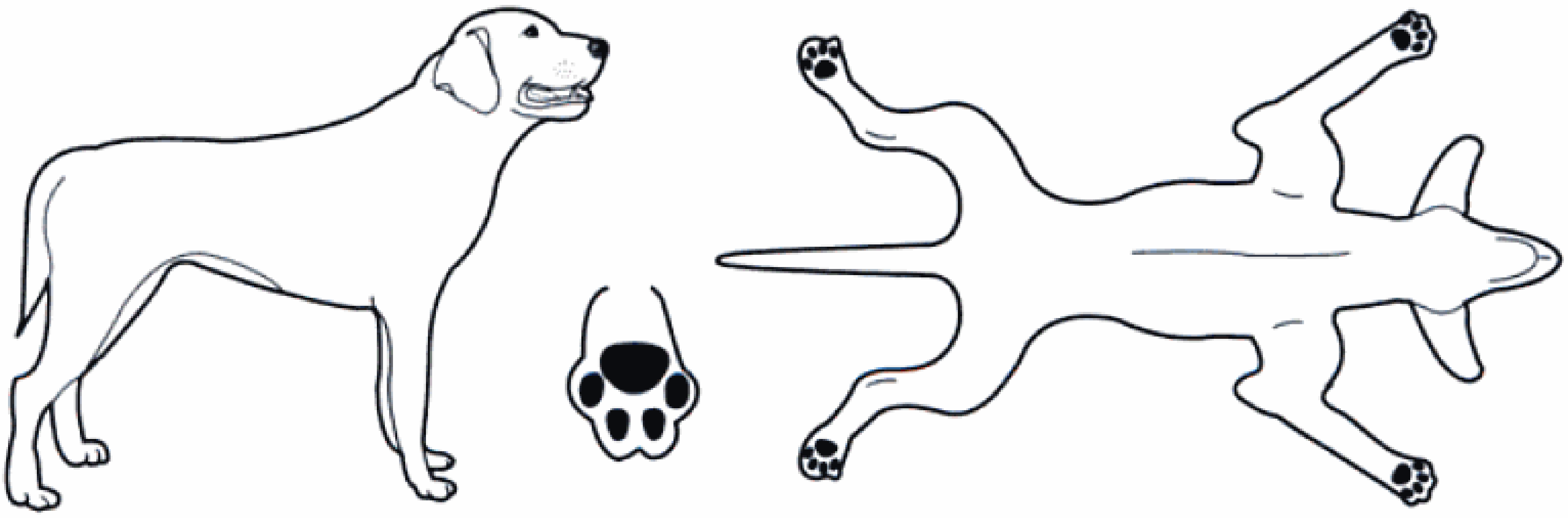
Aetiology: Unknown, but may be related to photoperiod and melatonin secretion from the pineal gland causing an exaggerated shedding. Most common in Boxers, English bulldogs and Airedale terriers but can be seen in other breeds.

Predominant lesions:

Well circumscribed patches of alopecia and hyperpigmentation that may wax and wane with the seasons or become permanent.

Lesion distribution:

Flanks, lateral chest.



Appropriate diagnostic tests:

Trichogram
Biopsy

Follicular dysplasia

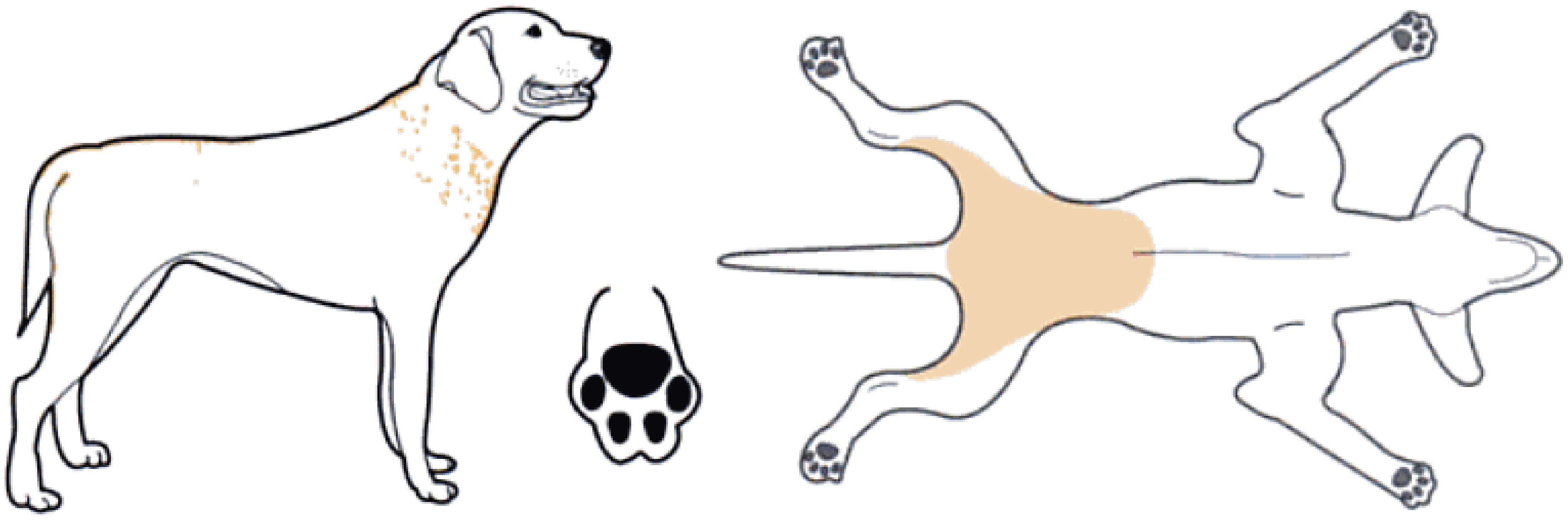
Aetiology: A genetically determined abnormality in hair follicle conformation leading to production of defective hair. Seen in Irish water spaniels, Portuguese water dogs, Curly coated retrievers, Siberian Huskies and occasionally in other breeds.

Predominant lesions:

Alopecia.

Lesion distribution:

Caudal dorsum, caudal thighs, perineum, neck but can affect the entire trunk.



Appropriate diagnostic tests:

Trichogram
Biopsy

Colour dilution alopecia and Black hair follicular dysplasia

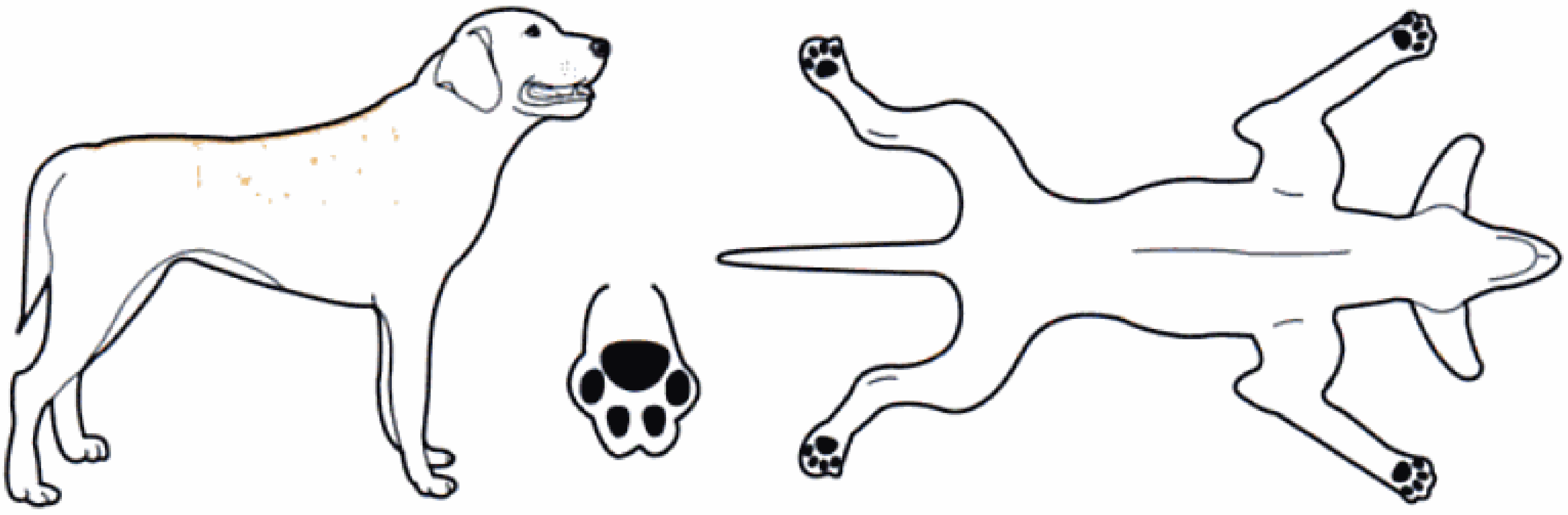
Aetiology: Genetically determined diseases in which the alopecia results from abnormal packaging of melanin granules within the hair shaft resulting in distortion and fracture. Colour dilution alopecia usually occurs in blue or fawn Doberman pinschers, Dachshunds, Great Danes, Whippets and occasionally in other breeds. Black hair follicular dysplasia occurs in dogs with bi- or tri-colour coats in which only the black hairs are affected.

Predominant lesions:

Alopecia, secondary staphylococcal pyoderma.

Lesion distribution:

Over the dorsum but may generalise, or be restricted to the black areas.



Appropriate diagnostic tests:

Trichogram
Biopsy

Pattern baldness

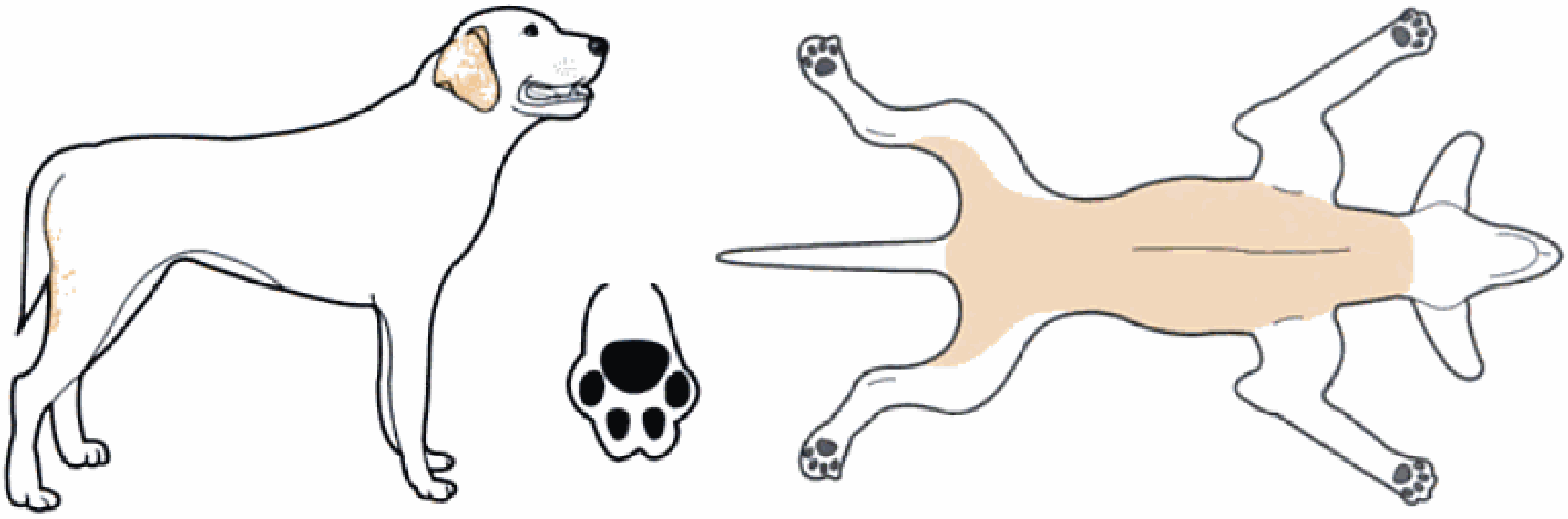
Aetiology: Genetically determined alopecia thought to be caused by miniaturisation of hair follicles. Seen most commonly in Dachshunds and Greyhounds.

Predominant lesions:

Alopecia.

Lesion distribution:

Pinnae, ventrum and caudo-medial thighs in Dachshunds; caudal thighs in Greyhounds.



Appropriate diagnostic tests:

Trichogram
Biopsy

Congenital alopecia

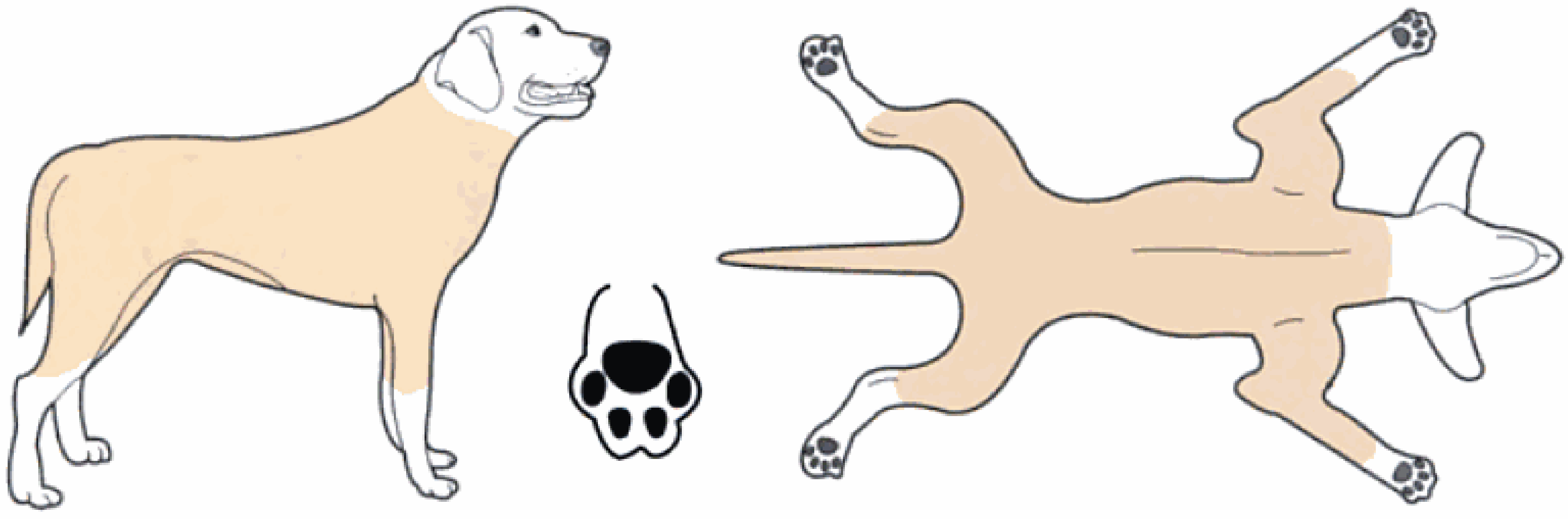
Aetiology: Genetically determined alopecia present at, or shortly after, birth caused by malformation of the hair follicles. Recognised as normal in the Chinese crested dog, Mexican hairless dog and the Sphinx cat.

Predominant lesions:

Alopecia, comedones, scaling and secondary infection.

Lesion distribution:

Generalised, or may spare the head and feet.



Appropriate diagnostic tests:

Trichogram
Biopsy

Telogen/Anagen defluxion

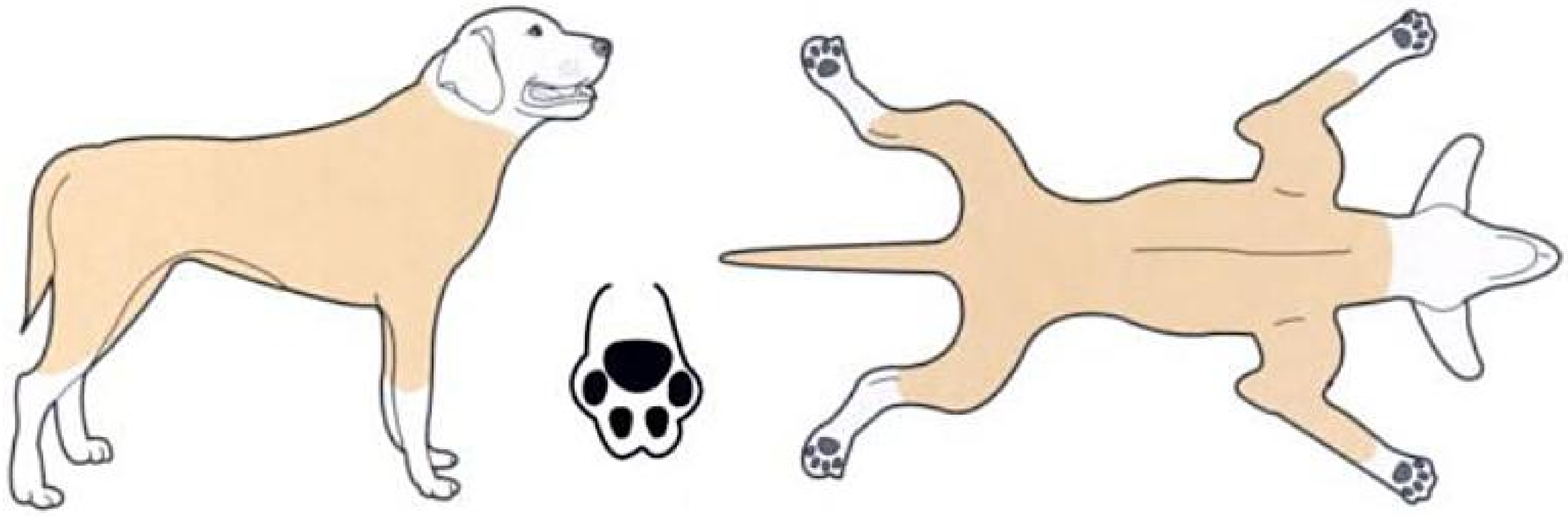
Aetiology: Telogen defluxion occurs when large numbers of hair follicles are synchronised in the telogen phase during a period of endocrine disease, stress, illness, pregnancy or lactation. When the endocrine disease is treated, the illness or stress resolves, or the lactation is over, the hair follicles return to anagen. The new hairs push the old hairs out resulting in a temporary but dramatic shed. Anagen defluxion usually occurs when the growth of anagen hairs is halted by chemotherapeutic drugs. However, alopecia is only seen in breeds with anagen dominated hair coats (Poodle, Bichon Frise, etc.).

Predominant lesions:

Excessive shedding, alopecia.

Lesion distribution:

Generalised.



Appropriate diagnostic tests:

Trichogram
Biopsy

Sebaceous adenitis

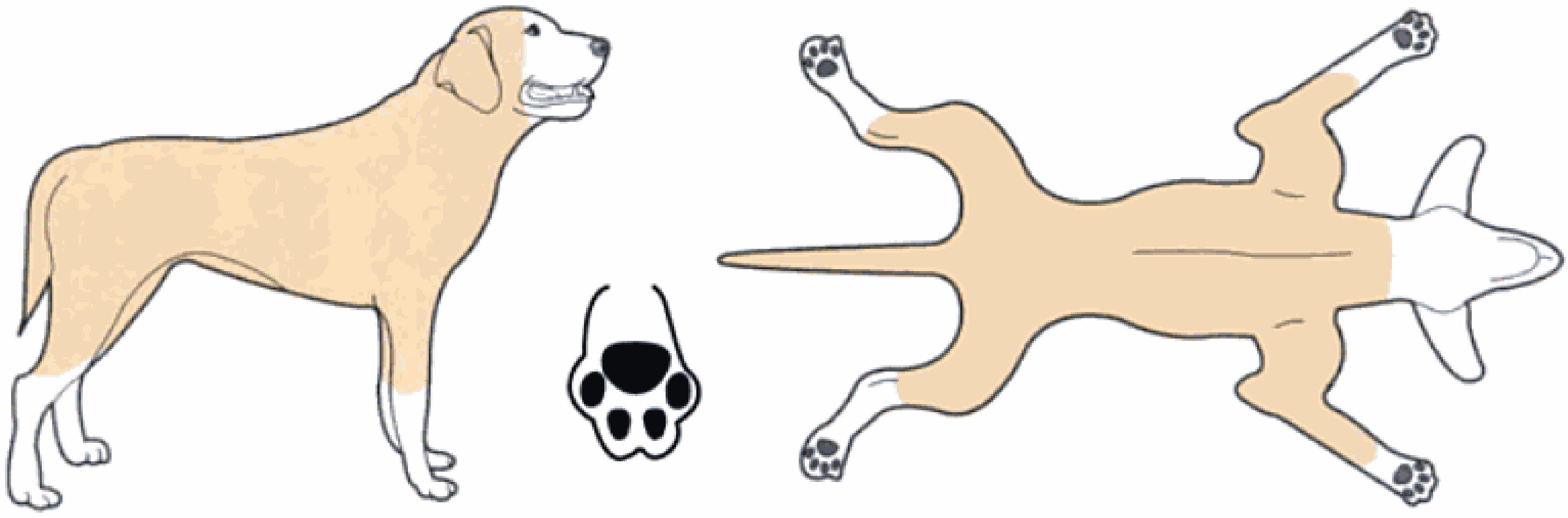
Aetiology: Granulomatous inflammation of the sebaceous glands of unknown cause, resulting in their destruction. Usually affects black Standard Poodles, Samoyeds, Japanese Akitas, Hungarian Vizslas and English Springer Spaniels.

Predominant lesions:

Alopecia and fine, silvery-white scaling.

Lesion distribution:

Usually generalised but can be multi-focal in Vizslas.



Appropriate diagnostic tests:

Trichogram
Biopsy

Feline paraneoplastic alopecia

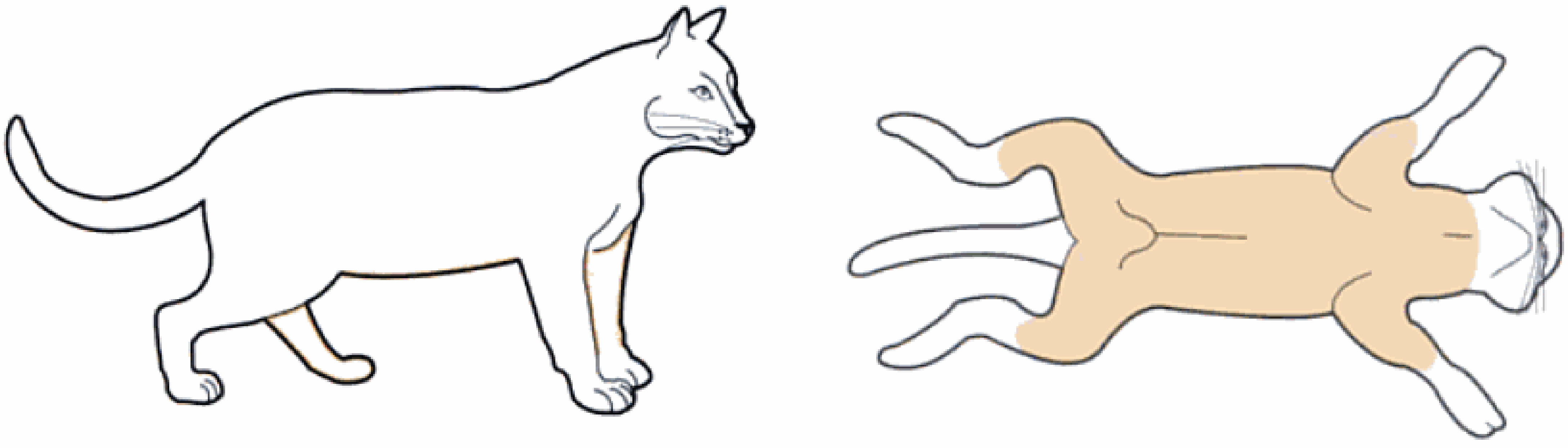
Aetiology: A sudden onset symmetrical alopecia seen in old cats associated with pancreatic adenocarcinomas.

Predominant lesions:

Alopecia associated with smooth, glistening skin.

Lesion distribution:

Ventrum and legs.



Appropriate diagnostic tests:

Biochemistry
Biopsy
Abdominal ultrasound
Exploratory laparotomy

Feline symmetrical alopecia

Aetiology: Feline symmetrical alopecia is most commonly due to pruritus (see Chapter 4). Rarely, it may be due to non-pruritic alopecia or psychogenic causes. In such cases, the differential diagnosis includes hyperadrenocorticism, hyperthyroidism, diabetes mellitus, paraneoplastic alopecia, telogen or anagen defluxion, lower urinary tract disease or psychogenic alopecia.

Predominant lesions and distribution:

See Chapter 4.

Appropriate diagnostic tests:

Trichogram
Biopsy
Haematology
Biochemistry
Adrenal function tests

Epitheliotropic lymphoma

Epitheliotropic lymphoma is a rare cause of alopecia that may or may not be pruritic. It is described in more detail in Chapter 4.

General diagnostic approach to alopecia

When approaching the problem of alopecia, it is important to note the similarities in many of the diseases described above under each presenting pattern. For example, staphylococcal pyoderma, demodicosis and dermatophytosis may all look the same on clinical examination. In the dog, staphylococcal infection and demodicosis would be the most likely causes of this pattern, whereas in cats, dermatophytosis would be more likely. Some of the more generalised alopecias such as endocrine diseases and follicular dysplasias can also appear remarkably similar.

Hence, as with pruritus, it is important to adopt a logical diagnostic approach that systematically rules out the various possibilities. Figures 5.1, 5.2 and 5.3 show diagnostic algorithms for localised, multi-focal and symmetrical alopecia respectively. As for pruritus, the clinician should superimpose their clinical experience on the approach suggested in these flow-charts.

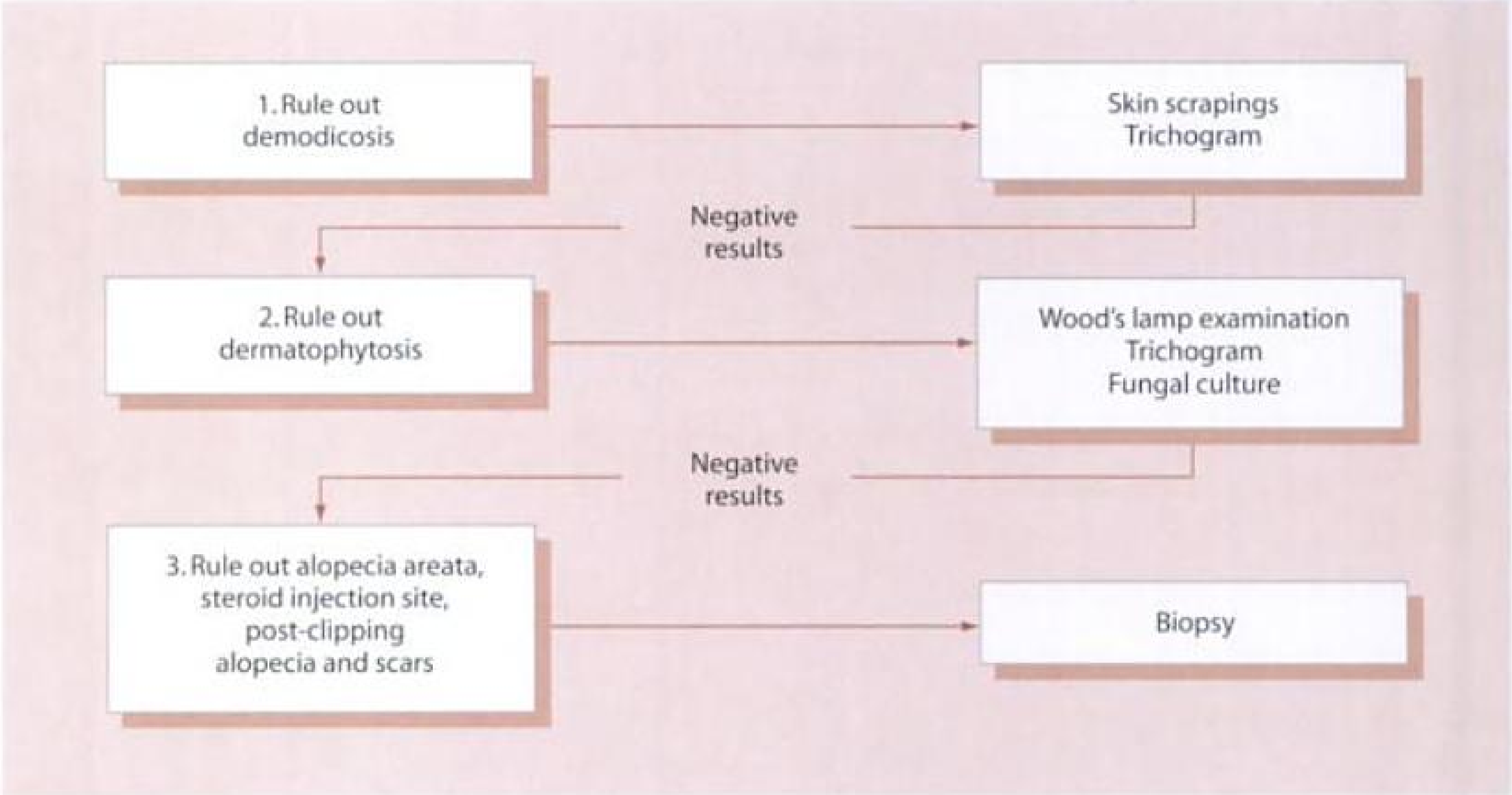


Figure 5.1 General diagnostic approach to localised alopecia.

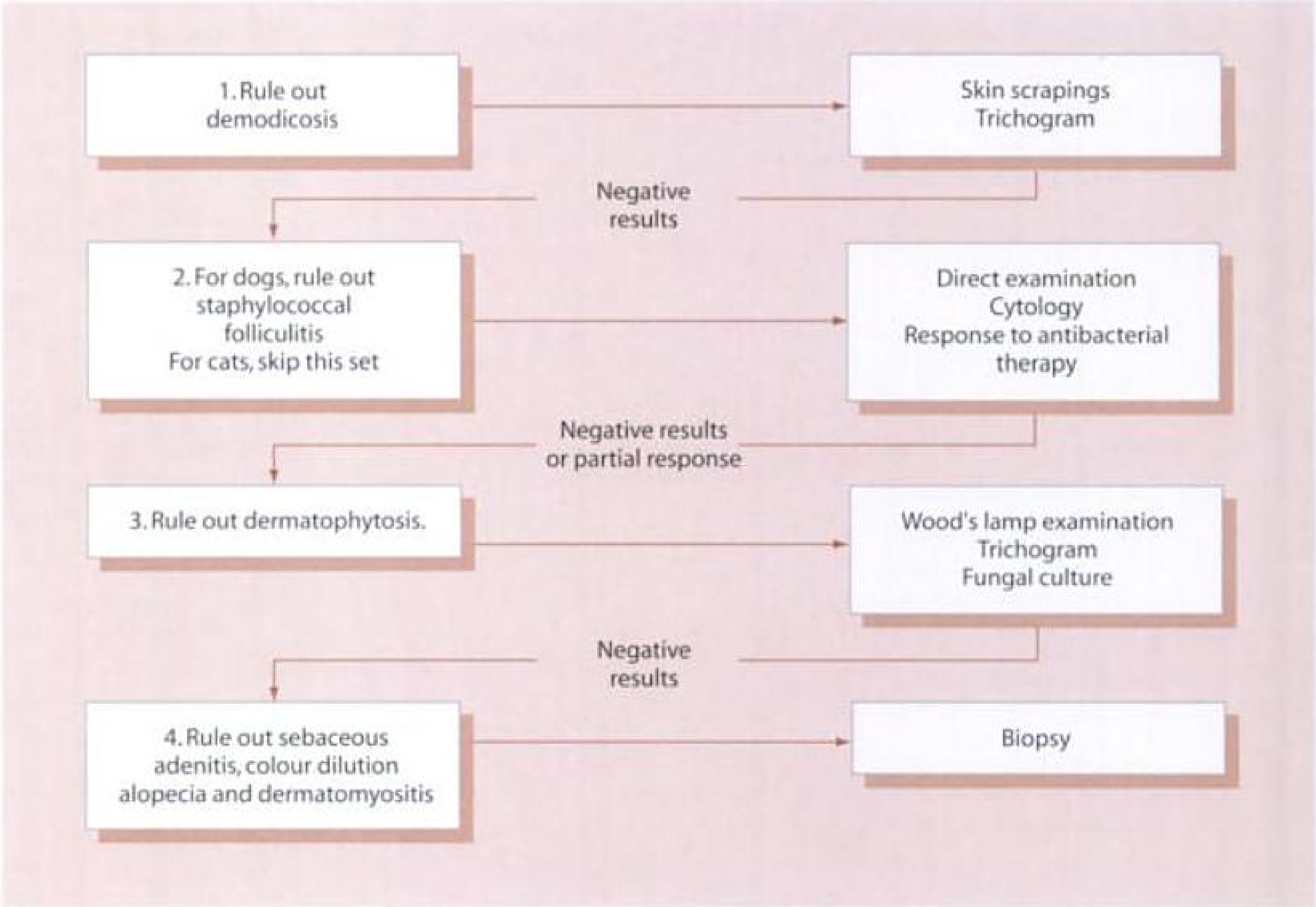

1. Rule out demodicosis
Skin scrapings
Trichogram
Negative results
2. For dogs, rule out staphylococcal folliculitis
For cats, skip this set
Direct examination
Cytology
Response to antibacterial therapy
Negative results or partial response
3. Rule out dermatophytosis.
Wood's lamp examination
Trichogram
Fungal culture
Negative results
4. Rule out sebaceous adenitis, colour dilution alopecia and dermatomyositis
Biopsy

Figure 5.2 General diagnostic approach to multi-focal patches of alopecia.

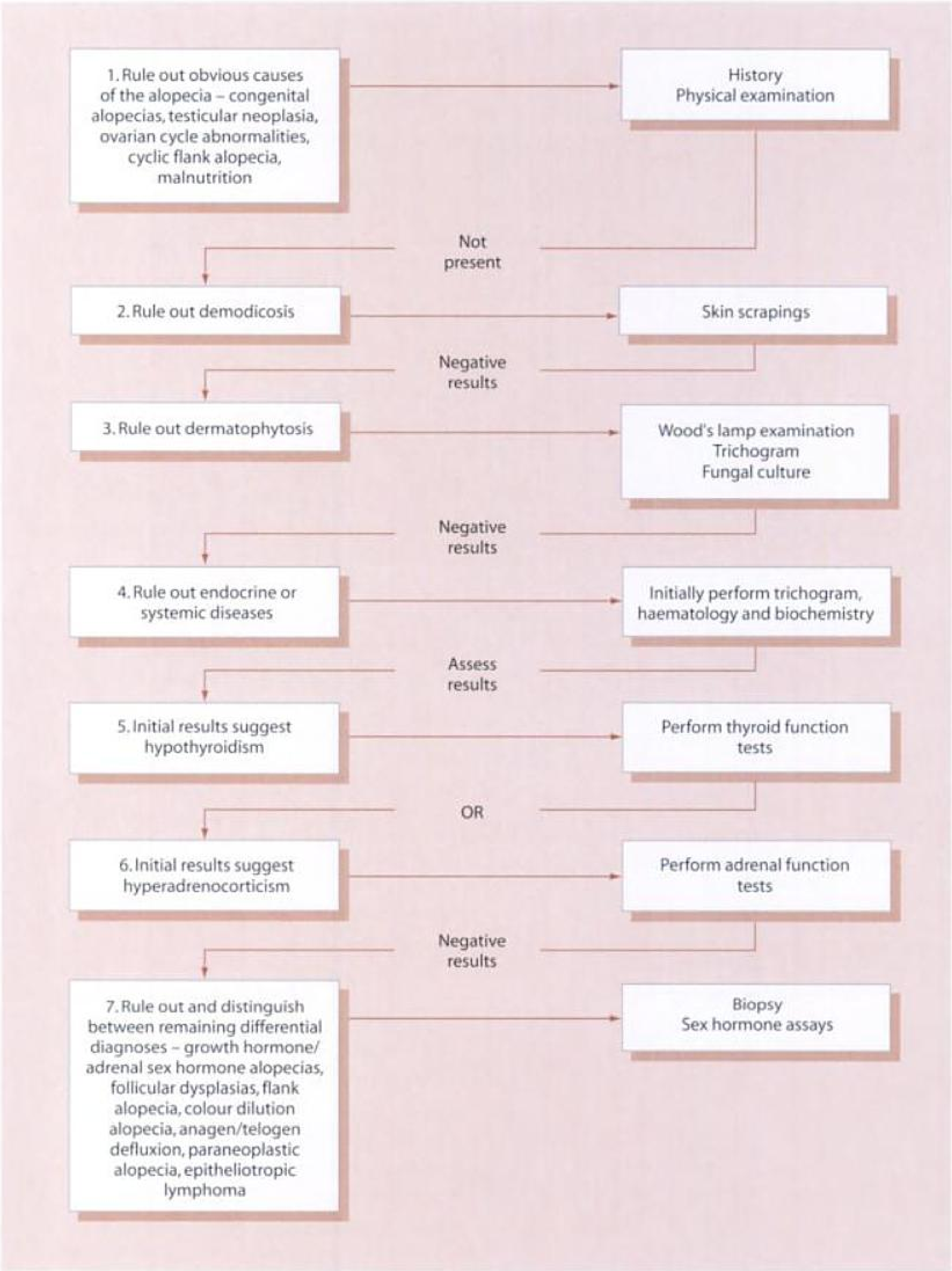

1. Rule out obvious causes of the alopecia – congenital alopecias, testicular neoplasia, ovarian cycle abnormalities, cyclic flank alopecia, malnutrition
History
Physical examination
Not present
2. Rule out demodicosis
Skin scrapings
Negative results
3. Rule out dermatophytosis
Wood's lamp examination
Trichogram
Fungal culture
Negative results
4. Rule out endocrine or systemic diseases
Initially perform trichogram, haematology and biochemistry
Assess results
5. Initial results suggest hypothyroidism
Perform thyroid function tests
OR
6. Initial results suggest hyperadrenocorticism
Perform adrenal function tests
Negative results
7. Rule out and distinguish between remaining differential diagnoses – growth hormone/adrenal sex hormone alopecias, follicular dysplasias, flank alopecia, colour dilution alopecia, anagen/telogen defluxion, paraneoplastic alopecia, epitheliotropic lymphoma
Biopsy
Sex hormone assays

Figure 5.3 General diagnostic approach to symmetrical or diffuse alopecia.

6

Scaling, crusting and seborrhoea

Differential diagnosis

Excessive skin scaling or seborrhoea is a common presenting dermatological problem in dogs and cats. The clinical signs are due to excessive production or abnormal formation of the stratum corneum, with or without a concurrent abnormality in skin lipid production. Many of the diseases mentioned in Chapters 4 and 5 can lead to excessive scaling, resulting in so-called secondary seborrhoea. In addition, there are a number of specific disorders in which scaling or seborrhoea is the main problem. These primary scaling disorders are often genetically determined and therefore have marked breed predilections.

When approaching the problem of excessive scaling, it is important to rule out the possible secondary causes first. These can be divided into those conditions that are pruritic (Table 6.1) and those that are non-pruritic (Table 6.2). Table 6.3 lists the primary scaling disorders with their breed predispositions.

Clinical features of scaling disorders

Secondary causes of pruritic seborrhoea

The clinical features of all the secondary causes of pruritic seborrhoea have been described in Chapter 4. If the animal is pruritic, the clinician should investigate this first.

Table 6.1 Diseases that may lead to pruritic seborrhoea.

Aetiological category	Specific disease
Ectoparasites	**Flea bite hypersensitivity (D and C)**
	Sarcoptes scabiei infestation (D)
	Notoedres cati infestation (C)
	Cheyletiella infestation (D and C)
Infectious agents	**Staphylococcal pyoderma (D)**
	Malassezia dermatitis (D)
Allergies	**Atopic dermatitis (D and C)**
	Food intolerance (D and C)
Neoplasia	Epitheliotropic lymphoma (D and C)

Those in bold would be most common in general practice. D = those diseases seen in dogs, C = those diseases seen in cats.

Table 6.2 Diseases that may lead to non-pruritic seborrhoea.

Aetiological category	Specific disease
Hormonal	**Hypothyroidism (D)**
	Hyperadrenocorticism (D and C)
	GH/adrenal sex hormone abnormalities (D)
Ectoparasites	**Demodicosis (D and C)**
	Cheyletiellosis (D and C)
Infections	**Dermatophytosis (D and C)**
	Leishmaniasis (D)
	FeLV associated dermatosis (C)
	FIV-associated dermatosis (C)
	Canine distemper virus infection (D)
Immune-mediated	Pemphigus foliaceus (D and C)
	Pemphigus erythematosus (D and C)
	Cutaneous lupus (D and C)
	Drug eruption (D and C)
Neoplasia	Epitheliotropic lymphoma (D and C)
Metabolic	Hepatocutaneous syndrome (D and C)
	Liver or renal disease (D and C)
Nutritional	Malnutrition or malabsorption (D and C)
Paraneoplastic	Thymoma-associated exfoliative dermatosis (C)
Environmental	Low humidity/dry heat (D)
	Poor grooming (C)

Those in bold would be most common in general practice. D = those diseases seen in dogs, C = those diseases seen in cats.

Secondary causes of non-pruritic seborrhoea

The clinical features of hypothyroidism, hyperadrenocorticism, sex hormone abnormalities, demodicosis, and dermatophytosis have been described in Chapter 5. In these diseases, the primary problem is usually alopecia. Epitheliotropic lymphoma, which may or may not be pruritic, is described in Chapter 4. Pemphigus foliaceus and pemphigus erythematosus usually cause crusting secondary to pustular eruptions and are described in Chapter 7. Cutaneous lupus usually causes ulceration as described in Chapter 8 but an exfoliative form is described here.

In this chapter, the clinical features of diseases that present primarily with scaling abnormalities will be described.

Leishmaniasis

Aetiology: A protozoal infection spread by sandflies and caused by a number of Leishmania species. Very rare in the UK, occasionally seen in the USA, but common in the Mediterranean basin and South America.

Predominant lesions:

Exfoliative dermatitis with silvery white, asbestos-like scaling; naso-digital hyperkeratosis. For details of the many systemic signs, see other texts.

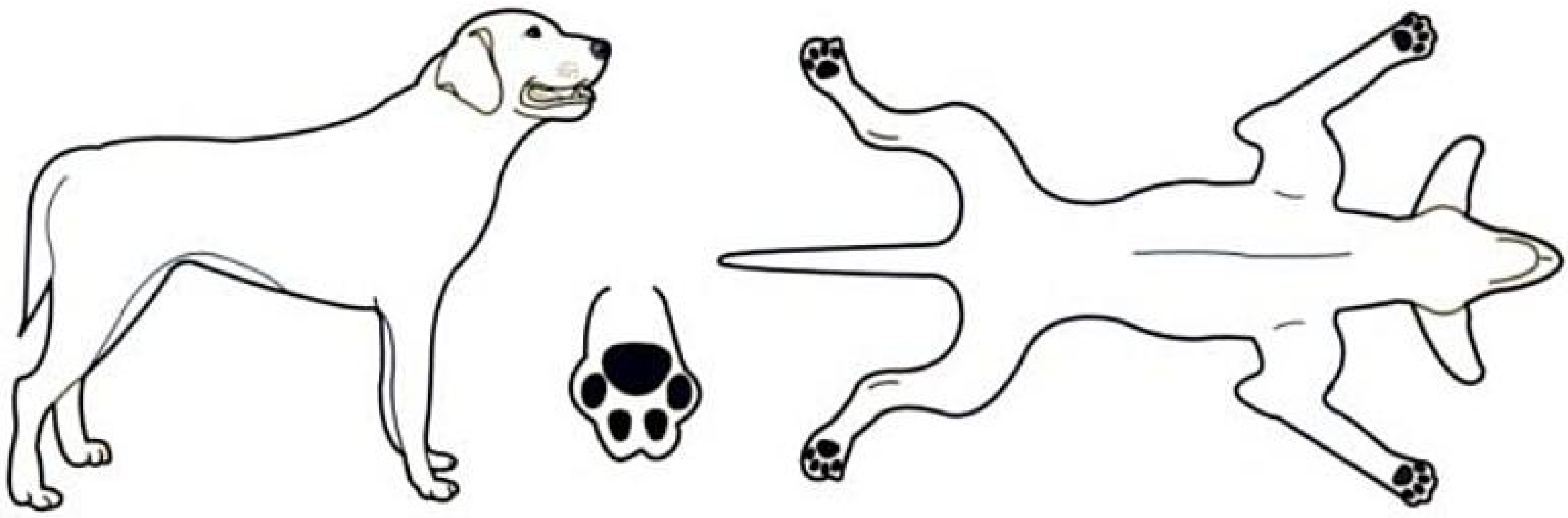
Table 6.3 Primary scaling disorders with breed predispositions.

Aetiological category	Specific disease	Affected breed
Functional epithelial abnormality (cornification defect)	**Primary seborrhoea**	**Cocker Spaniel** **Springer Spaniel** **Cavalier King Charles Spaniel** **Irish Setter** **Doberman Pinscher** West Highland White terrier Shar Pei
	Vitamin A responsive dermatosis	Cocker Spaniel
	Nasodigital hyperkeratosis	**Cocker Spaniel**
	Schnauzer comedone syndrome	Miniature Schnauzer
	Ichthyosis	WHW terrier
	Feline acne	**Cats**
	Feline tail gland hyperplasia	Cats
Nutritional/metabolic	Zinc responsive dermatosis	Siberian Husky Alaskan malamute
	Lethal acrodermatitis	English Bull Terrier
Immune-mediated	Sebaceous adenitis	Standard Poodle Japanese Akita Samoyed English Springer Spaniel Hungarian Vizsla

Those in bold would be most common in general practice. In many cases, the genetic association and pathogenesis have been assumed rather than proven.

Lesion distribution:

Usually head, pinnae and extremities but may generalise.



Appropriate diagnostic tests:

Serology
Cytology of bone marrow or lymph nodes (see other texts for details)
Biopsy

FeLV and FIV associated dermatoses

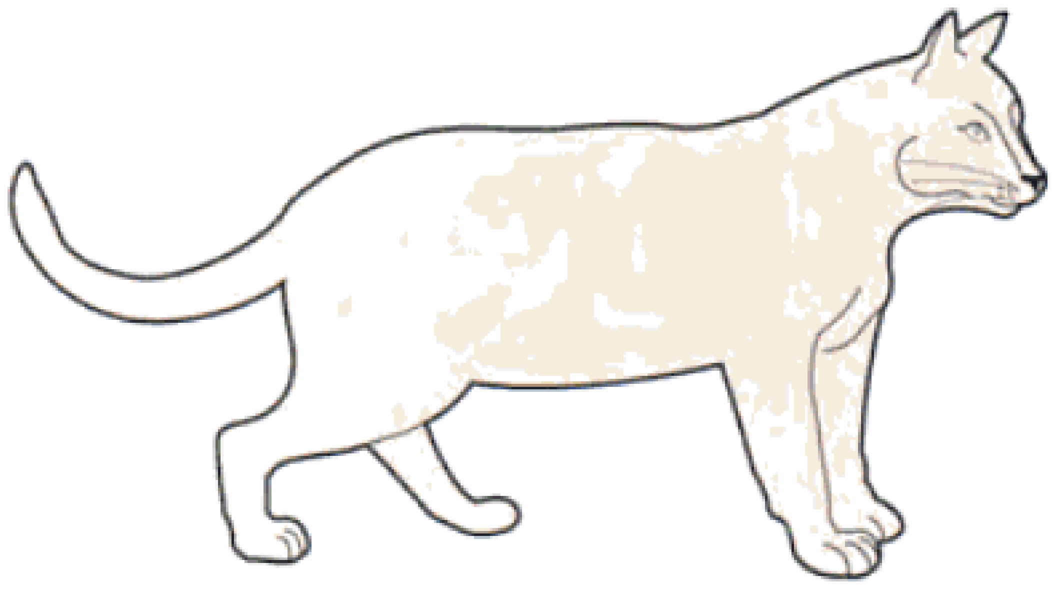
Aetiology: Very rare skin diseases caused by infection of keratinocytes with Feline leukaemia virus (proven) or Feline immunodeficiency virus (suspected).

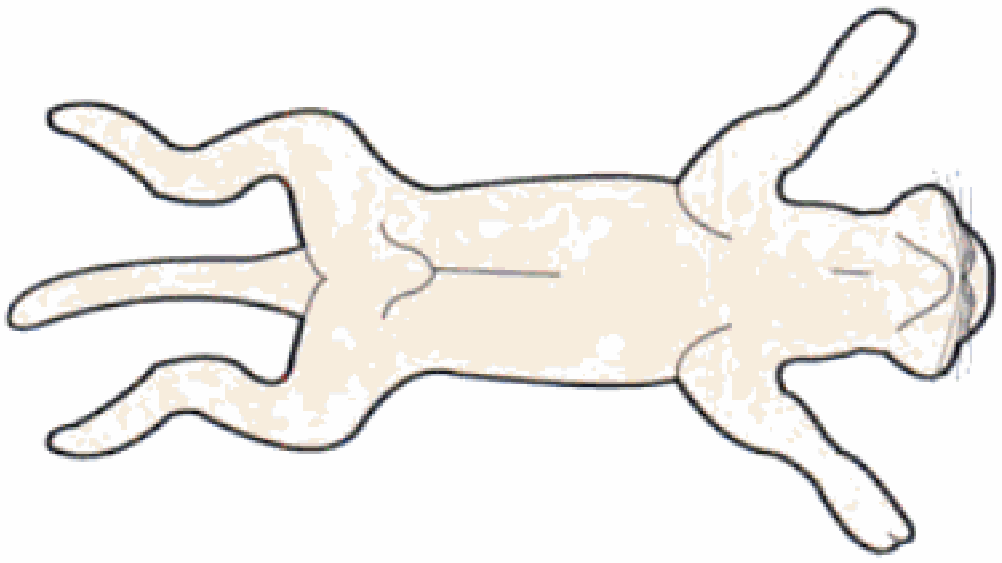
Predominant lesions:

Excessive scaling and crusting, alopecia, crusted papules.

Lesion distribution:

Both dermatoses usually start on the head but can become generalised.





Appropriate diagnostic tests:

FeLV serology
FIV serology
Biopsy

Canine distemper virus infection

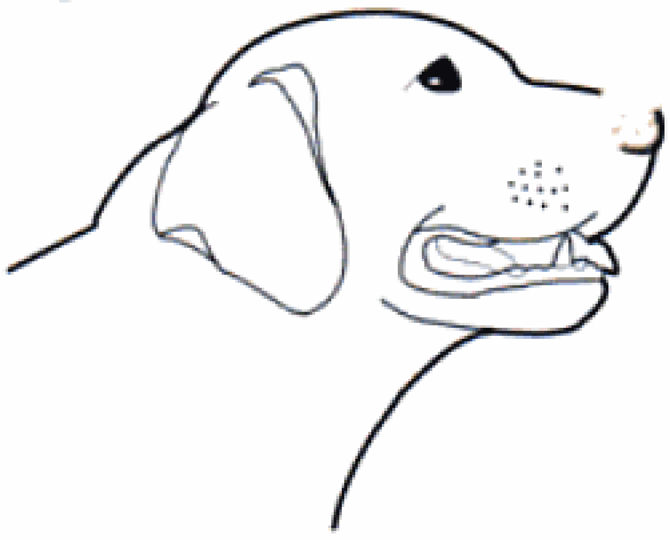
Aetiology: Infection of footpad and nasal planum keratinocytes with distemper virus. A rare disorder in areas where vaccine prophylaxis is routinely used.

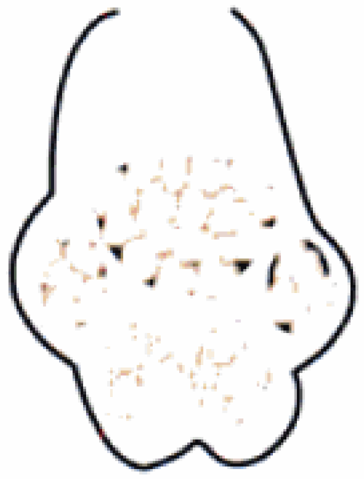
Predominant lesions:

Hyperkeratosis.

Lesion distribution:

Nasal planum and footpads.





Appropriate diagnostic tests:

Biopsy

Cutaneous lupus

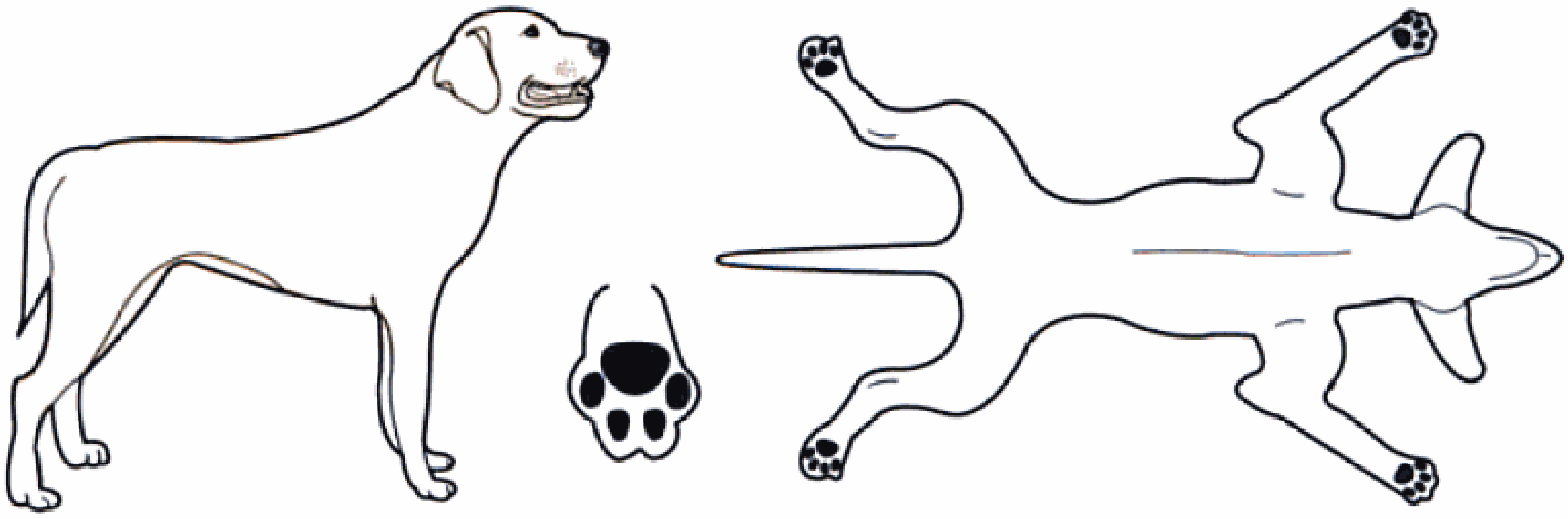
Aetiology: A rare disease caused by autoimmunity to various nuclear and cytoplasmic antigens, including DNA. Lupus most commonly presents as ulceration of the nasal planum or muco-cutaneous junctions (see Chapter 8). However, it can lead to generalised exfoliation. A breed specific form may exist in the German Shorthaired Pointer (lupoid dermatosis of the German Shorthaired Pointer).

Predominant lesions:

Scaling and exfoliation.

Lesion distribution:

Usually starts on the face, pinnae and trunk and then becomes generalised.



Appropriate diagnostic tests:

Haematology
Biochemistry
Urinalysis
Antinuclear antibody test
Biopsy

Drug eruption

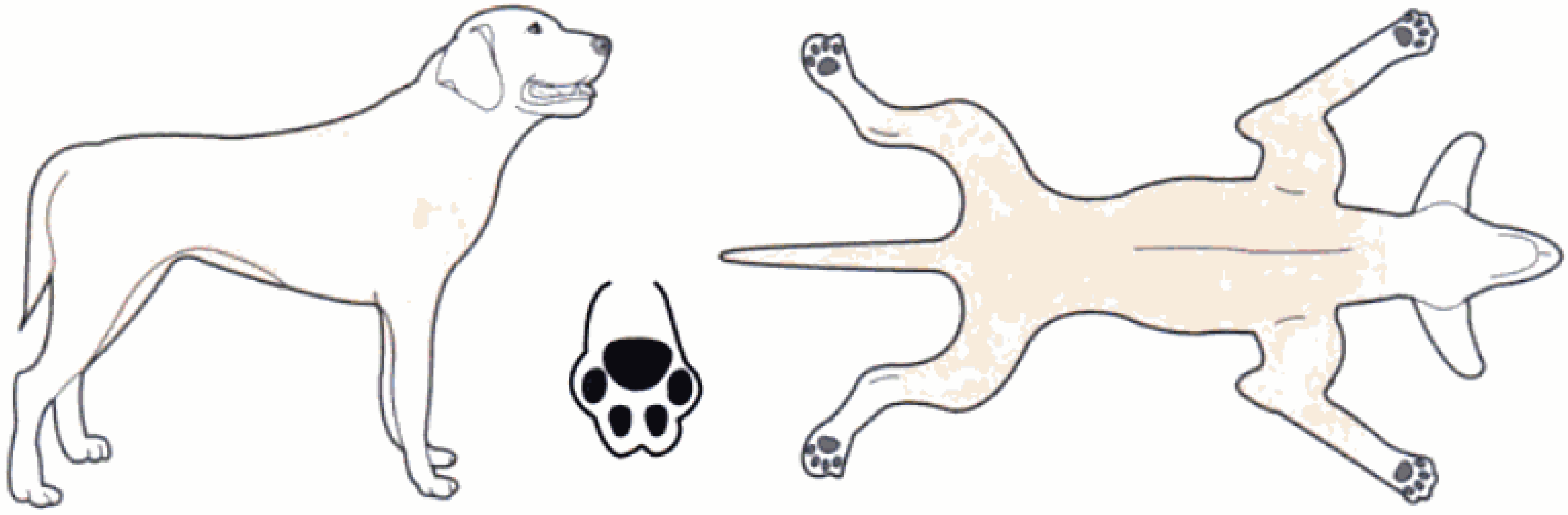
Aetiology: An adverse reaction to a drug leading to a cutaneous eruption. Drug eruptions occur sporadically, take many forms and will appear in other chapters of this book. One form is generalised exfoliation or exfoliative erythroderma.

Predominant lesions:

Exfoliation, erythroderma.

Lesion distribution:

Usually on the trunk but may be generalised.



Appropriate diagnostic tests:

Biopsy

Hepatocutaneous syndrome (superficial necrolytic dermatitis, metabolic epidermal necrosis, necrolytic migratory erythema)

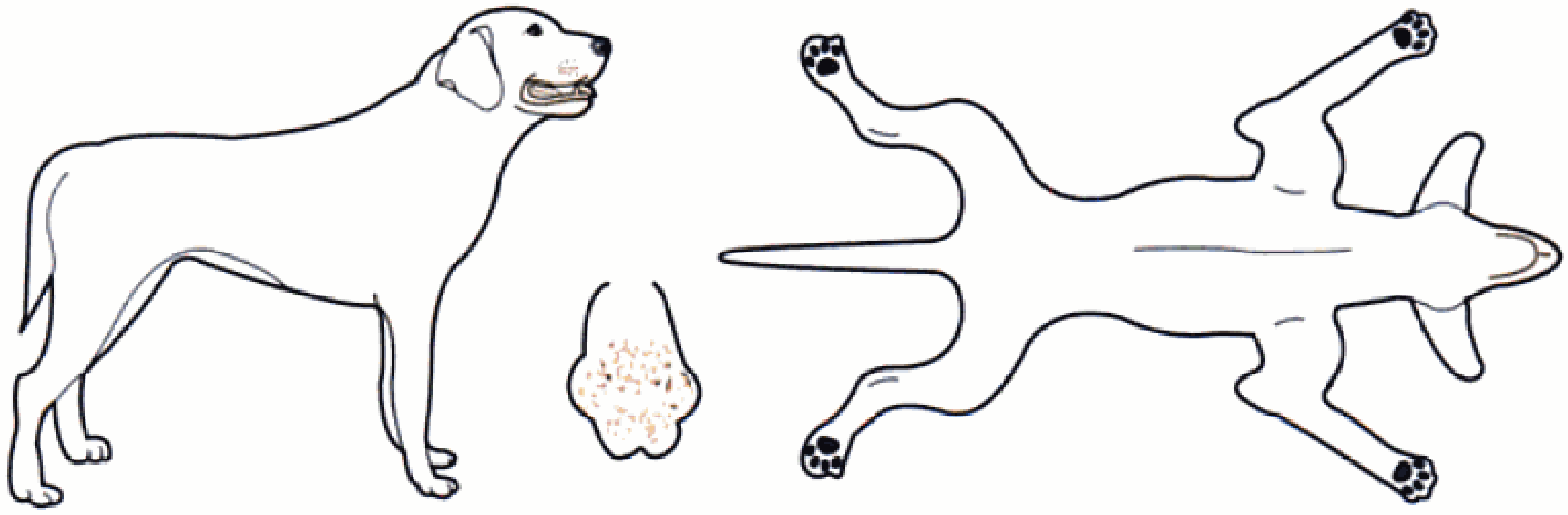
Aetiology: A rare skin disease usually seen in old dogs associated with liver pathology but also caused by glucagon-secreting pancreatic tumours. The skin changes in both cases may be due to cutaneous amino acid deficiency.

Predominant lesions:

Scaling, crusting and ulcers on the skin; hyperkeratosis and fissuring on the footpads.

Lesion distribution:

Muzzle, lips, eyelids, elbows, hocks, scrotum, ventrum, footpads.



Appropriate diagnostic tests:

Haematology
Biochemistry
Biopsy
Abdominal ultrasound

Liver and renal disease

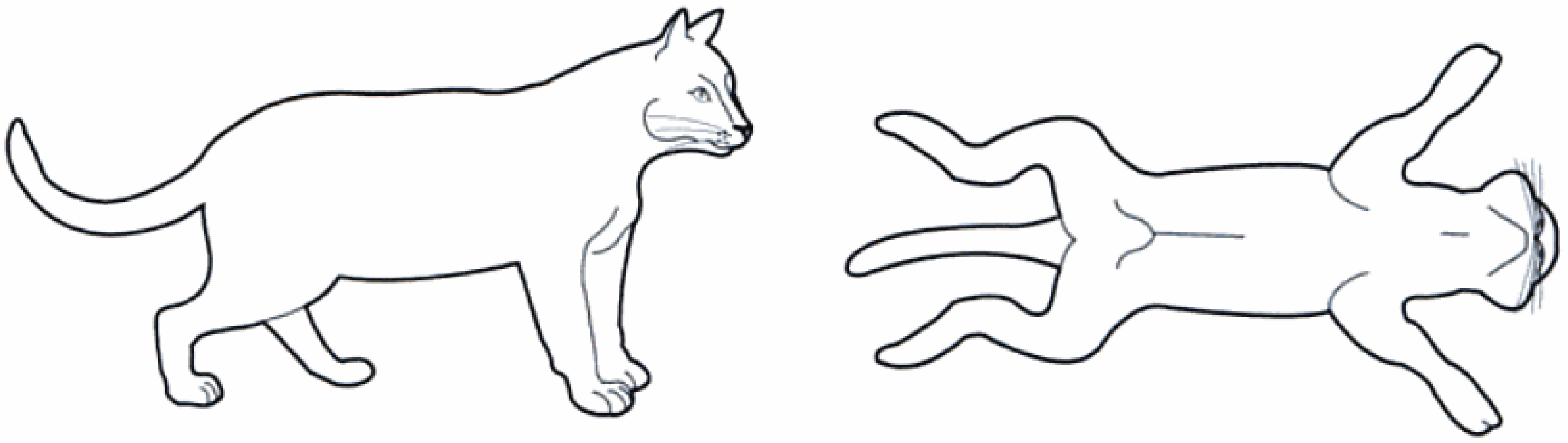
Aetiology: Excessive skin scaling due to the general debilitating effects of liver and renal failure.

Predominant lesions:

Scaling.

Lesion distribution:

Usually over the dorsum and trunk.



Appropriate diagnostic tests:

Haematology
Biochemistry

Malnutrition or malabsorption

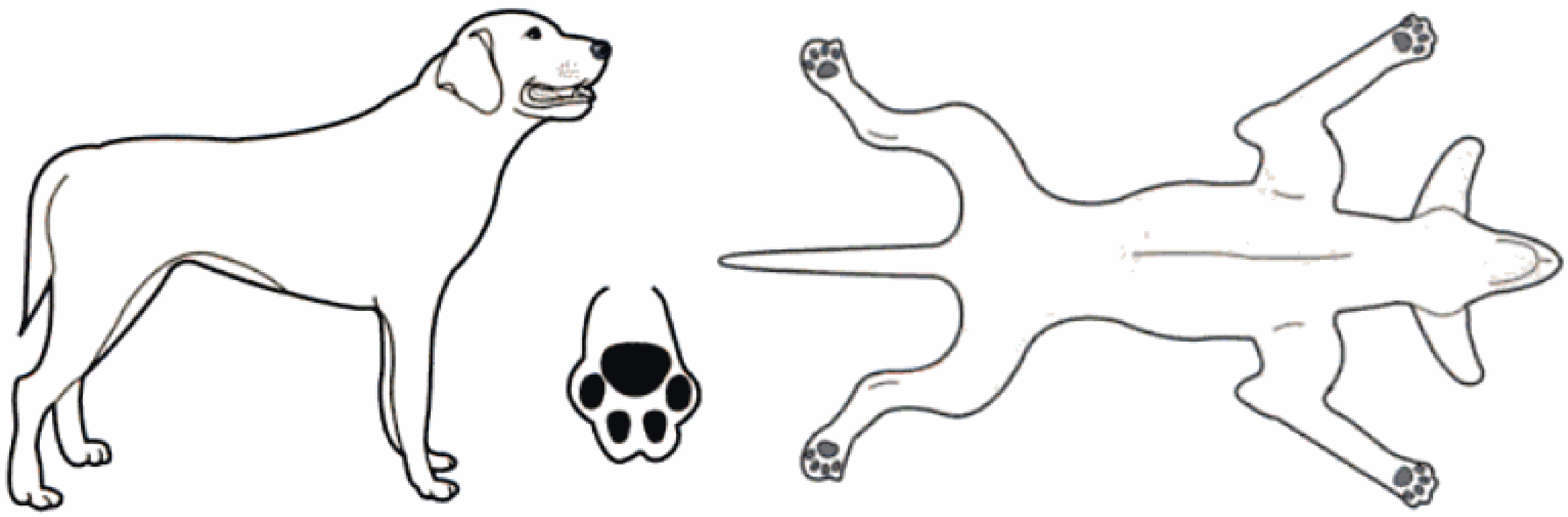
Aetiology: Cutaneous deficiency of protein, zinc, fat or essential fatty acids. Zinc deficiency can occur in young, rapidly growing large dogs fed on cereal diets. The syndrome is described in more detail below.

Predominant lesions:

Scaling.

Lesion distribution:

Generalised.



Appropriate diagnostic tests:

Response to dietary change
Gastrointestinal function tests
Biopsy

Thymoma-associated exfoliative dermatosis

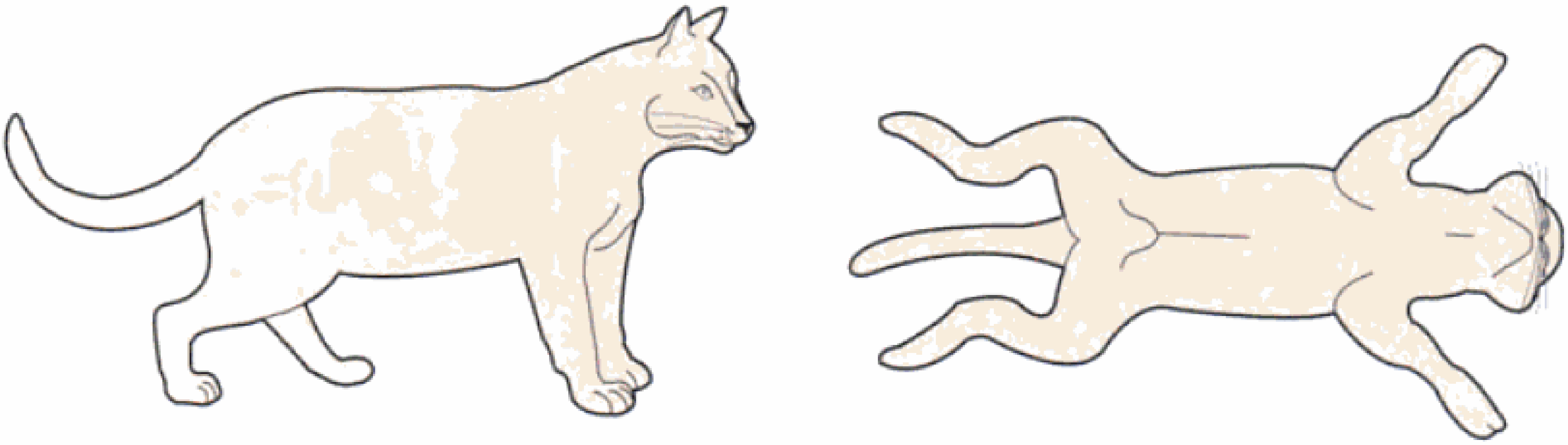
Aetiology: A rare paraneoplastic cutaneous disease seen in cats in association with thymic tumours.

Predominant lesions:

Scaling and exfoliation.

Lesion distribution:

Generalised.



Appropriate diagnostic tests:

Thoracic radiography
Biopsy

Low humidity/dry heat

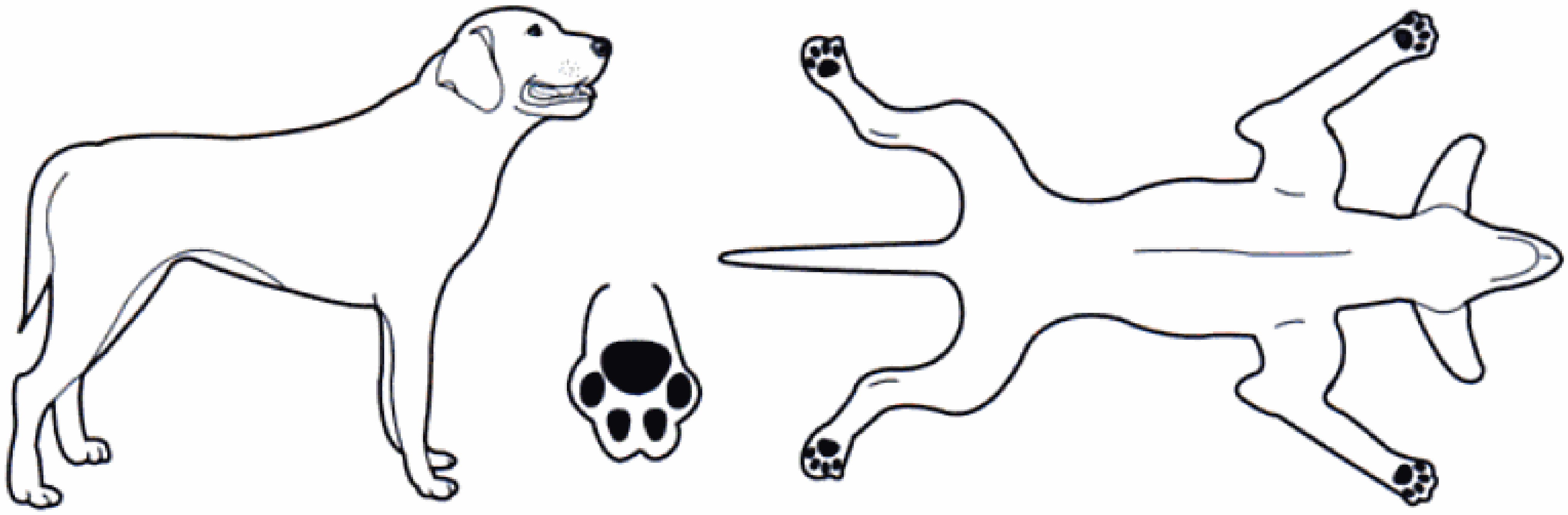
Aetiology: Excessive dryness of the skin due to inadequate environmental humidity.

Predominant lesions:

Mild scaling.

Lesion distribution:

Mainly over the dorsum.



Appropriate diagnostic tests:

Rule out of other causes

Poor grooming seborrhoea

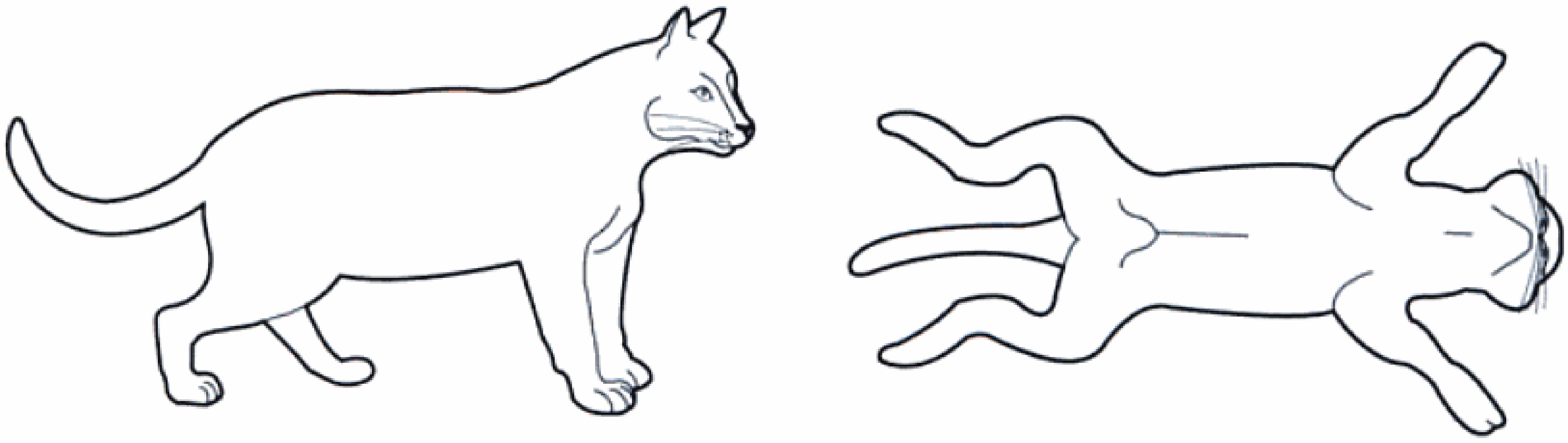
Aetiology: Excessive scaling associated with poor grooming, especially in elderly cats.

Predominant lesions:

Dry scaling.

Lesion distribution:

Mainly over the dorsum



Appropriate diagnostic tests:

Rule out of other causes, including musculoskeletal disease

Primary seborrhoea

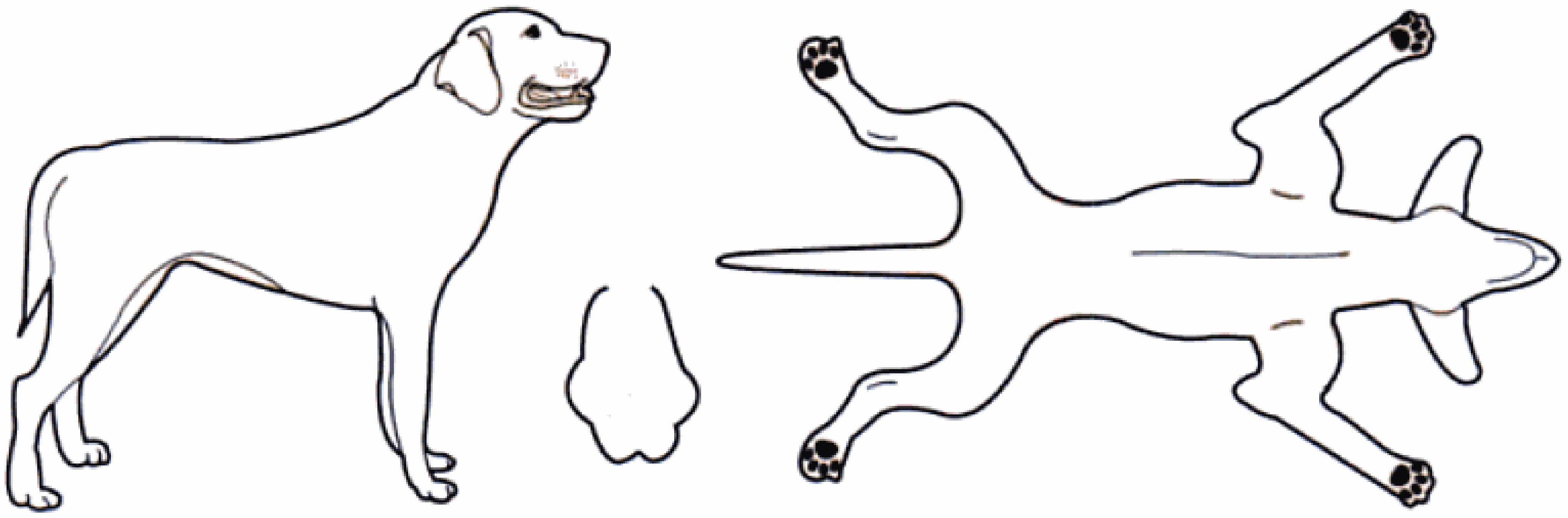
Aetiology: A genetically determined abnormality affecting the surface epidermis, follicular epithelium, nasal planum and footpads. In American Cocker Spaniels, the defect has been shown to be a hyperproliferation of the epithelium and sebaceous glands, resulting in accelerated and excessive production of the stratum corneum. Similar mechanisms are assumed in other breeds, but this has not been proven. The type of seborrhoea and its distribution varies depending on the breed affected.

Predominant lesions:

Cocker Spaniels, Springer Spaniels, Cavalier King Charles Spaniels, West Highland White Terriers, Shar Peis - Greasy scale (seborrhoea oleosa), comedones, follicular casts, otitis, secondary staphylococcal and Malassezia infections.

Lesion distribution:

Face, pinnae, ventral neck, interdigital skin, footpads, perineum, ventral abdomen, around nipples, axillae, in skin folds. Can be more generalised.

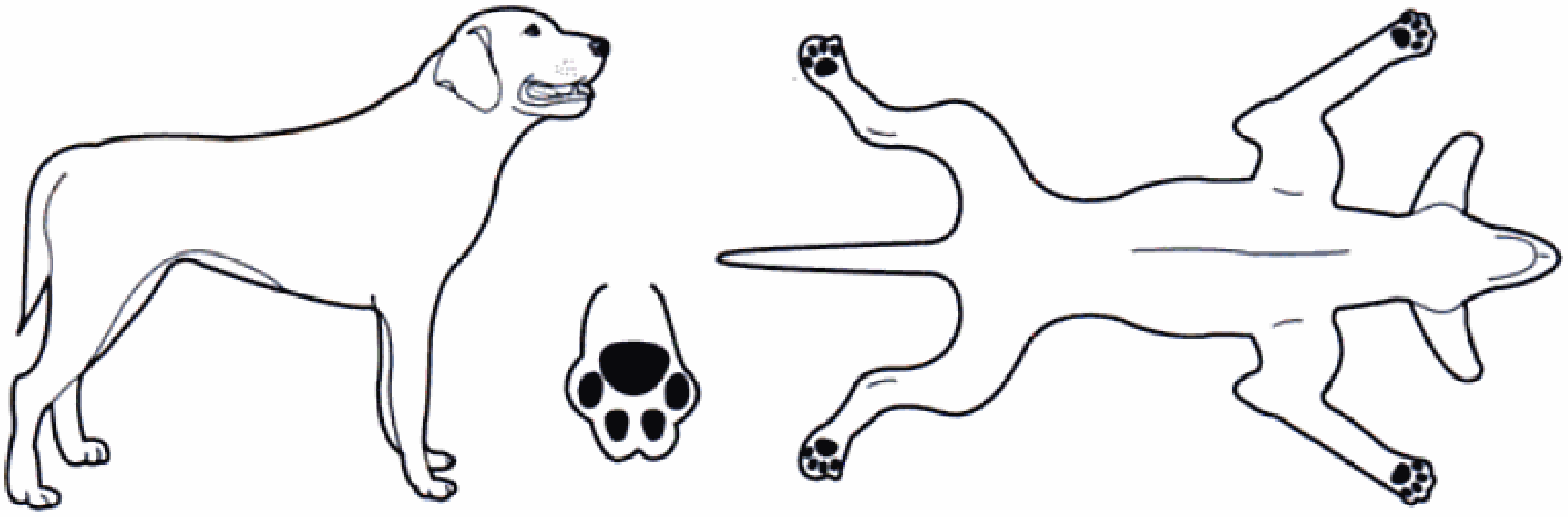


Predominant lesions:

Irish Setters, Doberman pinschers – Dry scaling (seborrhoea sicca).

Lesion distribution:

Over the dorsum and trunk.



Appropriate diagnostic tests:

Tests to rule out other causes
Biopsy

Vitamin A responsive dermatosis

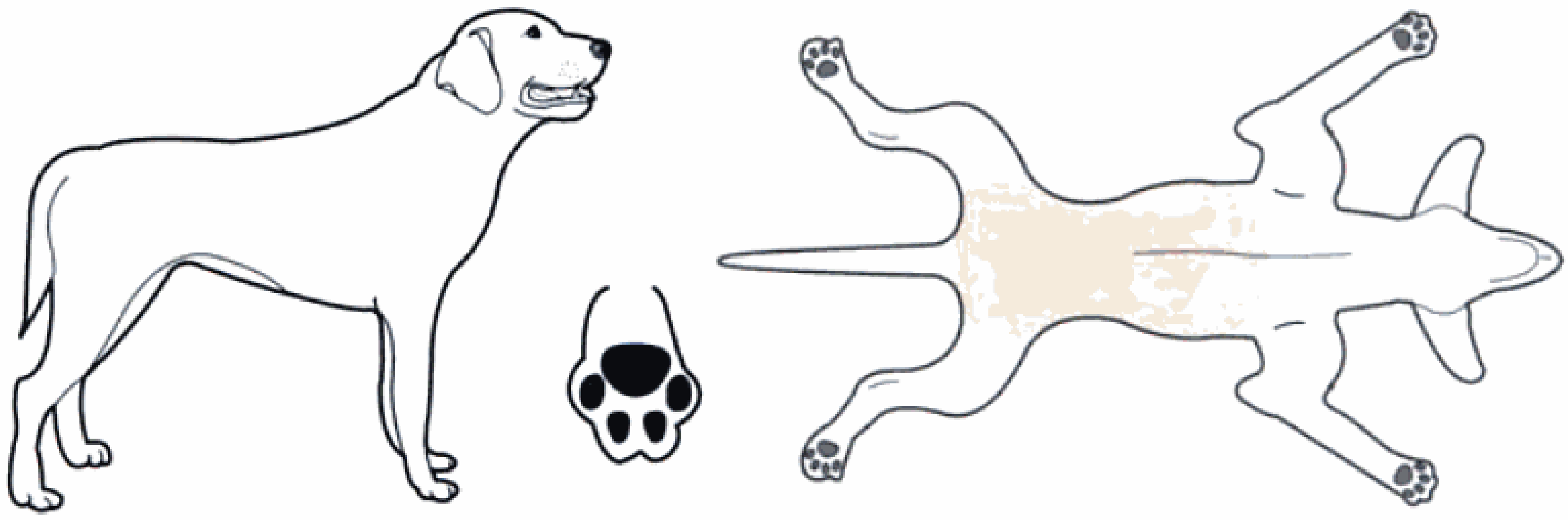
Aetiology: A form of cornification disorder that primarily affects the hair follicles. Usually seen in Cocker Spaniels.

Predominant lesions:

Large comedones and prominent follicular casts.

Lesion distribution:

Ventral chest and abdomen.



Appropriate diagnostic tests:

Biopsy

Naso-digital hyperkeratosis

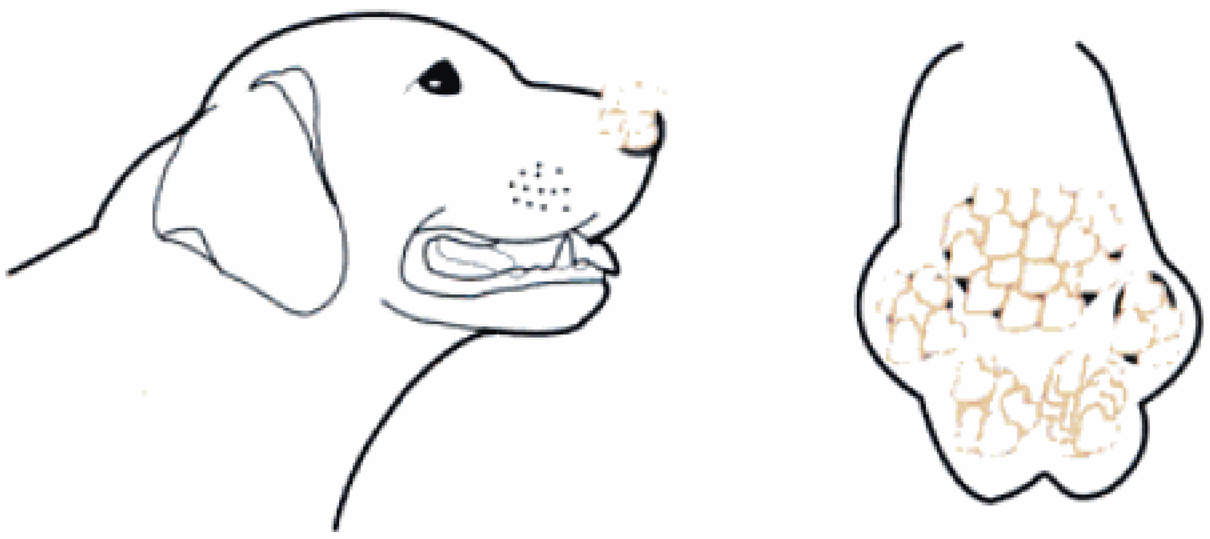
Aetiology: Hyperproliferation of the epithelium of the nose and footpads. Most commonly an idiopathic, age-related change but may also be seen in many of the diseases described in this chapter. Most common in Cocker Spaniels. A breed-specific form of footpad hyperkeratosis has been reported in Irish Setters and the Dogue de Bordeaux.

Predominant lesions:

Hyperkeratosis.

Lesion distribution:

Nasal planum and footpads.



Appropriate diagnostic tests:

Biopsy

Schnauzer comedone syndrome

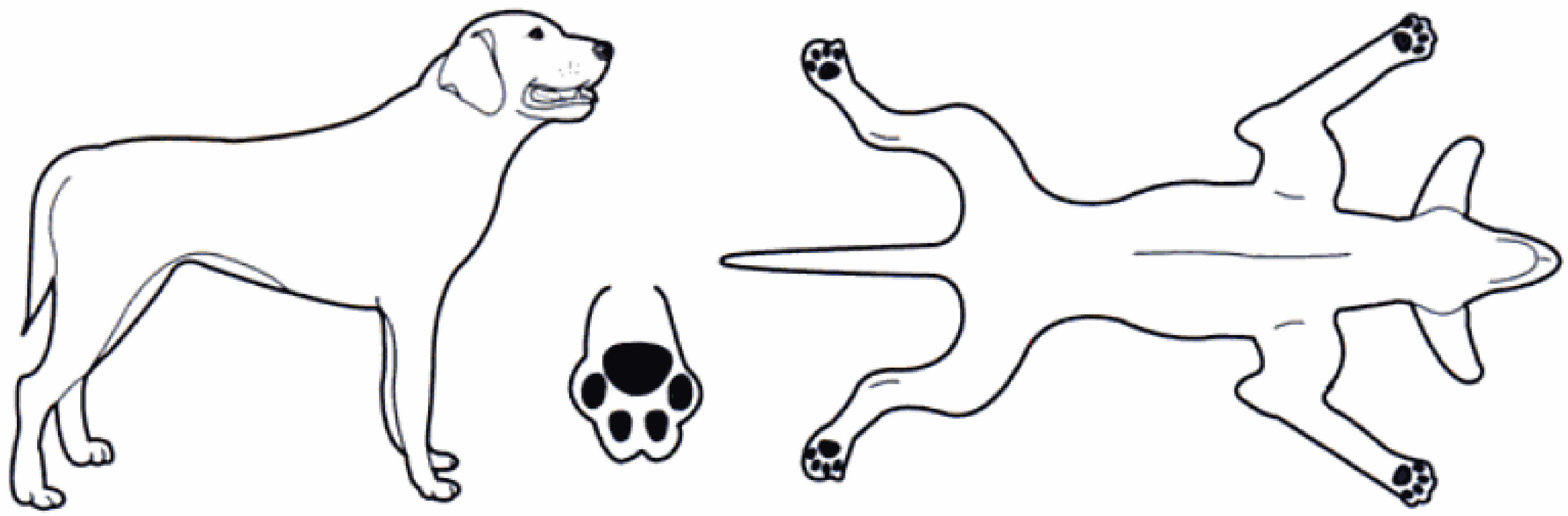
Aetiology: A genetically determined abnormality of the follicular epithelium.

Predominant lesions:

Comedones.

Lesion distribution:

Over the dorsum.



Appropriate diagnostic tests:

Biopsy

Ichthyosis

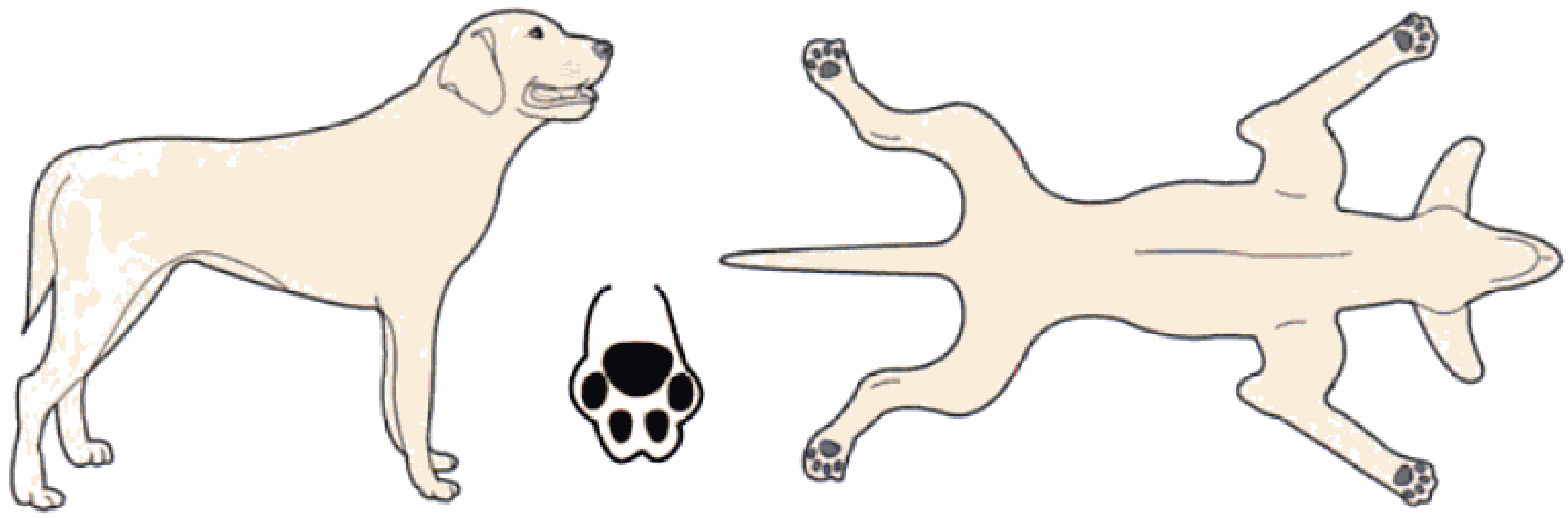
Aetiology: A very rare, genetically determined, congenital skin disease (lesions are present at birth) caused by massive overproduction of the stratum corneum.

Predominant lesions:

Tightly adherent, thick, verrucous scales which desquamate in large sheets.

Lesion distribution:

Generalised, including the footpads.



Appropriate diagnostic tests:

Biopsy

Feline acne

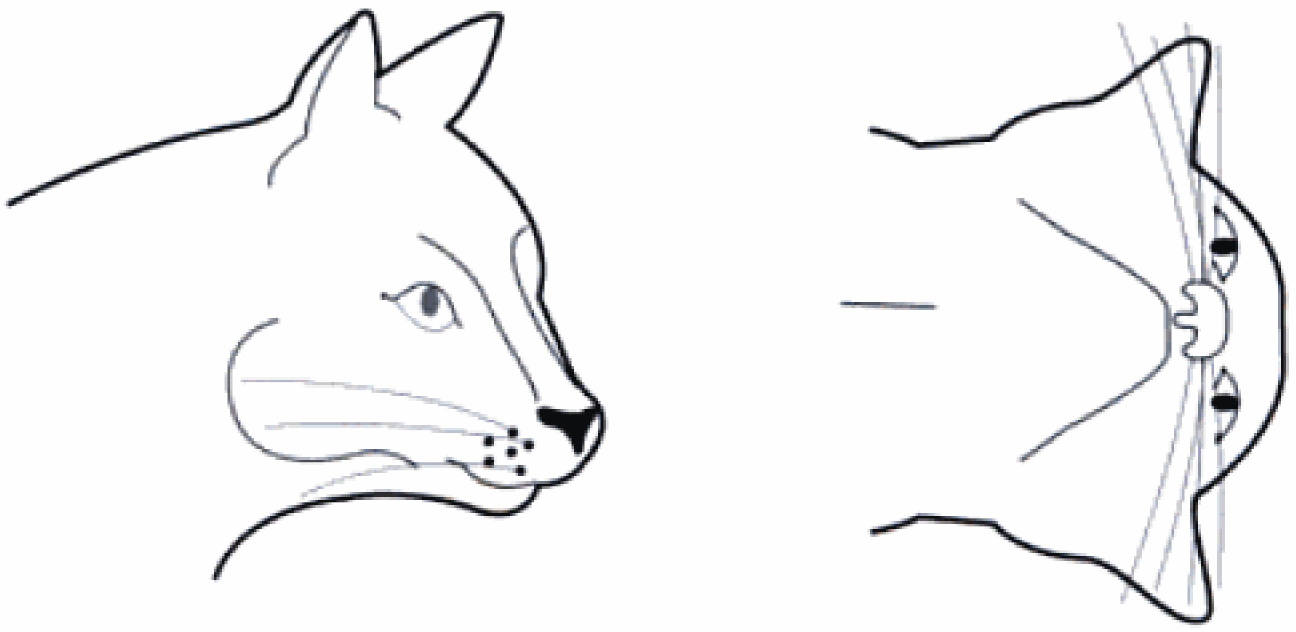
Aetiology: Usually an idiopathic disorder of follicular cornification, but can rarely be associated with demodicosis.

Predominant lesions:

Comedones and black, adherent debris. May develop secondary bacterial and Malassezia infections.

Lesion distribution:

Chin



Appropriate diagnostic tests:

Skin scraping
Cytology
Biopsy

Feline tail gland hyperplasia (stud tail)

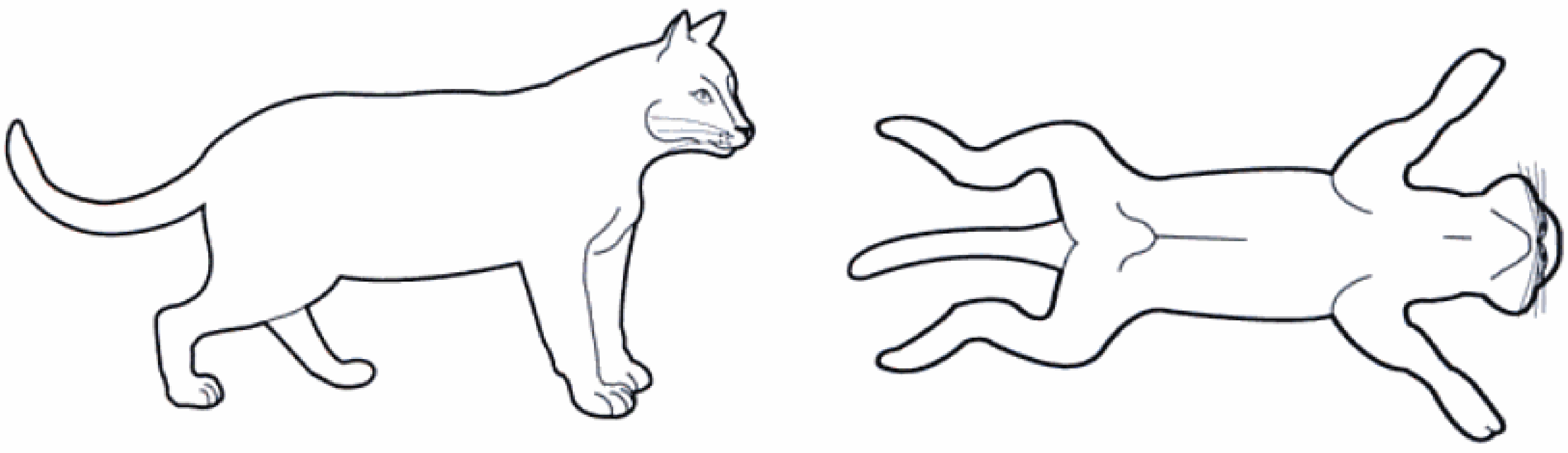
Aetiology: Hyperplasia of the sebaceous glands located along the dorsal aspect of the cat's tail (supracaudal organ). May be idiopathic or secondary to demodicosis and dermatophytosis.

Predominant lesions:

Greasy scales and crusts with matting of the overlying fur. May develop secondary bacterial and Malassezia infections.

Lesion distribution:

Dorsal tail.



Appropriate diagnostic tests:

Skin scraping
Cytology
Trichogram
Fungal culture
Biopsy

Zinc responsive dermatosis

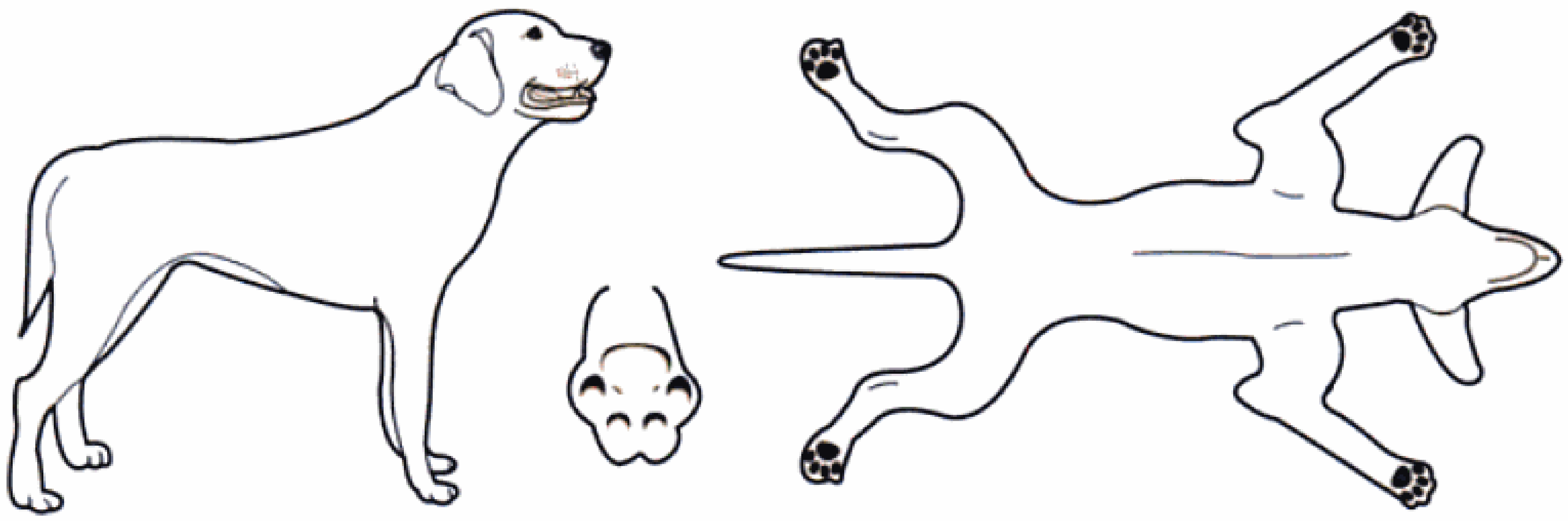
Aetiology: Two syndromes are recognised. Syndrome 1 is a genetically determined inability to adequately absorb zinc from the intestinal tract. It is seen in Siberian Huskies and Alaskan malamutes. Syndrome 2 is seen in rapidly growing dogs fed diets that contain high quantities of phytates or calcium (interfere with zinc absorption) or are deficient in zinc.

Predominant lesions:

Scaling, crusting, erythema and alopecia, with or without pruritus.

Lesion distribution:

Muzzle, chin, periocular, pinnae, ear canals, scrotum, prepuce, vulva, elbows, hocks, feet.



Appropriate diagnostic tests:

Cytology
Biopsy

Lethal acrodermatitis

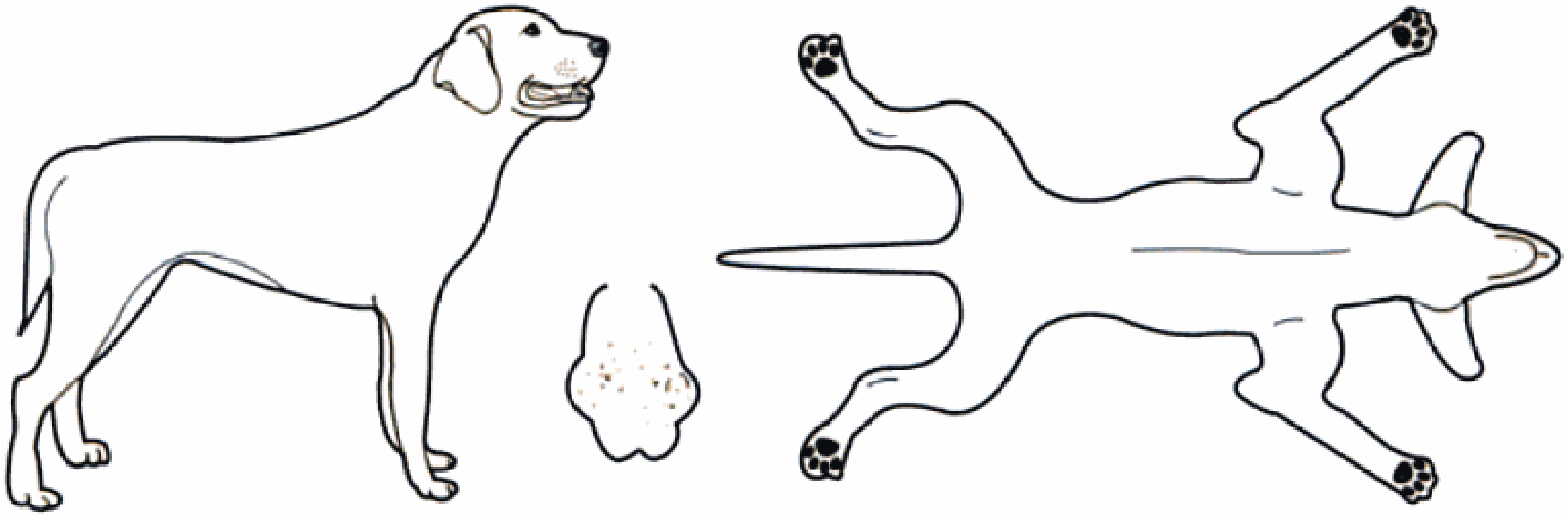
Aetiology: An inherited, autosomal recessive trait leading to defective metabolism of zinc and copper. It is a lethal disease seen only in English Bull terriers.

Predominant lesions:

Cutaneous scaling, crusting and erythema, fissuring of footpads. The dogs also have an abnormally arched hard palate, retarded growth, splayed feet, and may suffer from diarrhoea and chronic respiratory tract infections. The average survival time is 7 months.

Lesion distribution:

Face, feet, footpads, legs but may be more generalised. Secondary staphylococcal and Malassezia infections are common.



Appropriate diagnostic tests:

Cytology
Biopsy

Sebaceous adenitis

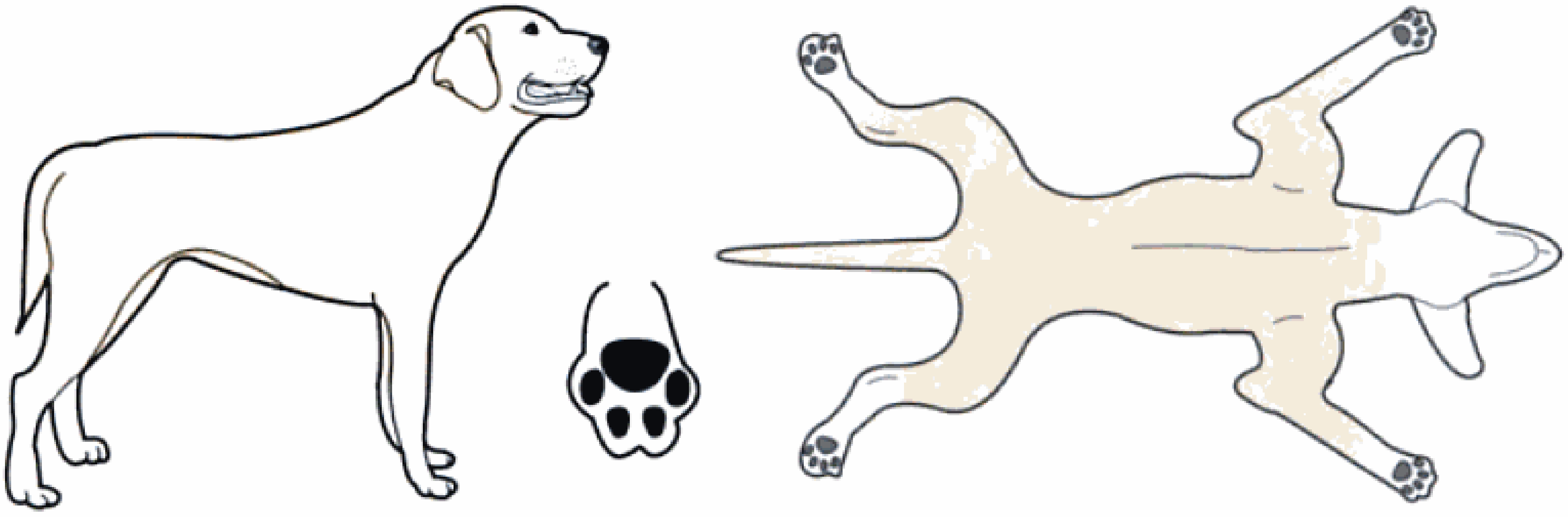
Aetiology: Granulomatous inflammation of the sebaceous glands of unknown cause, resulting in their destruction. Usually affects black Standard Poodles, Samoyeds, Japanese Akitas, Hungarian Vizslas and English Springer Spaniels. The condition causes both marked scaling and alopecia and is also described in Chapter 5.

Predominant lesions:

Alopecia and fine, silvery-white scaling. May also lead to greasy scaling.

Lesion distribution:

Usually generalised but can be multi-focal in Vizslas.



Appropriate diagnostic tests:

Trichogram
Biopsy

General diagnostic approach to scaling disorders

As with the problems of pruritus and alopecia, the history and clinical examination may suggest the most likely diagnosis in an animal with a scaling disorder. If this is the case, focused diagnostic tests can be performed to confirm the clinical suspicion. However, due to overlap between the clinical presentations of the diseases described above, it is often necessary to formulate a list of prioritised differential diagnoses. It is important when doing this not to assume that a particular breed is suffering from a primary scaling disorder. Although the breed may suggest that a primary scaling disorder is included on the differential diagnosis list, it does not preclude the possibility that the animal has another disease that is leading to secondary scaling. Hence, the golden rule when approaching this problem is to always consider the possibility of secondary causes before assuming an animal has a primary scaling disorder.

Figure 6.1 shows a diagnostic algorithm for scaling disorders. When using this algorithm, the clinician should use information obtained from the history and clinical examination to determine if all the steps are appropriate for a particular case. For example, it would not be necessary to rule out demodicosis or cheyletiellosis in a dog suffering only from footpad hyperkeratosis. It can be seen from this flow chart that skin biopsy is a very valuable tool when assessing scaling. Histopathological examination is the only way to definitively diagnose many of the diseases described in this chapter.

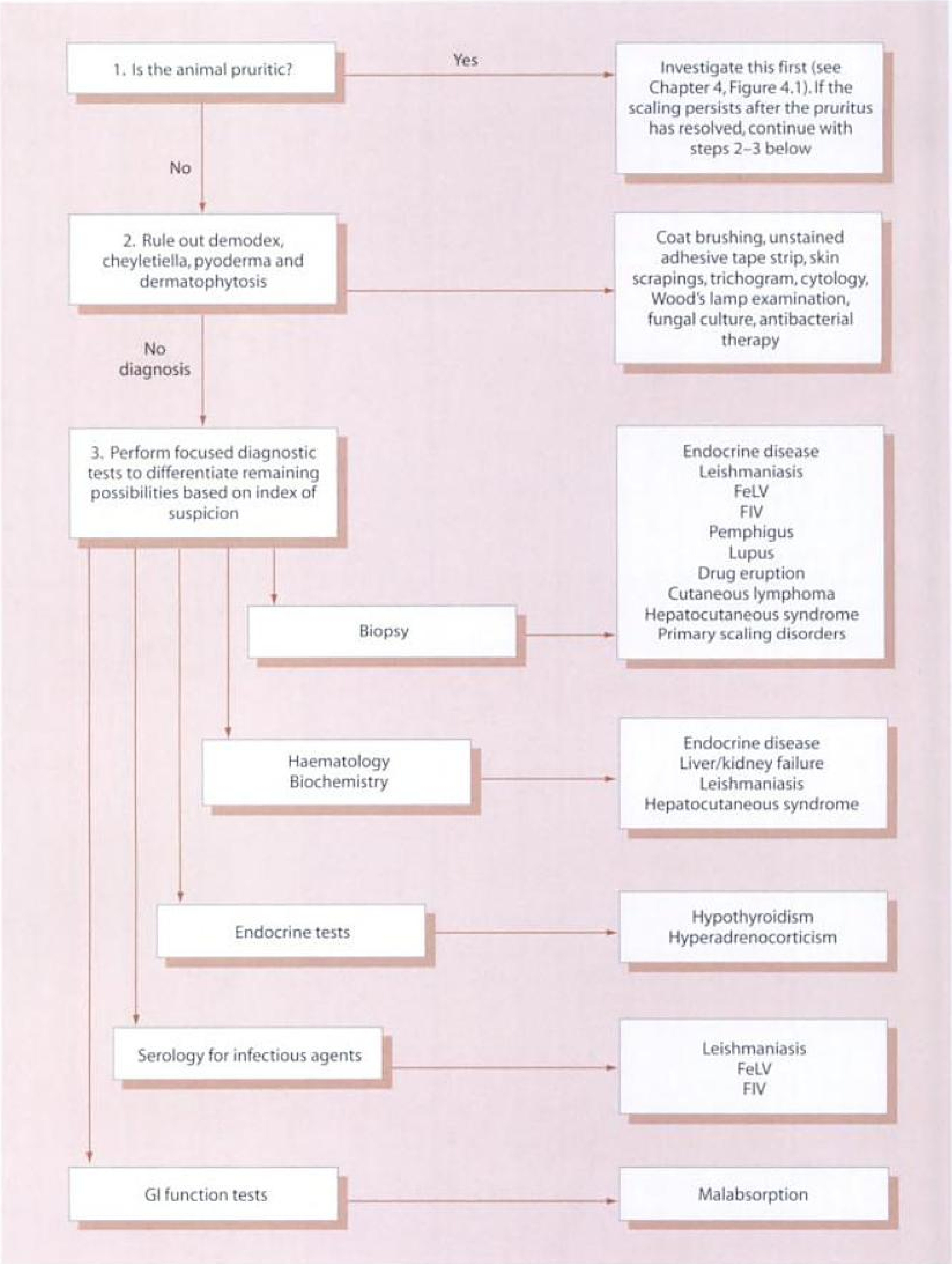

1. Is the animal pruritic?
Yes
Investigate this first (see Chapter 4, Figure 4.1). If the scaling persists after the pruritus has resolved, continue with steps 2–3 below
No
2. Rule out demodex, cheyletiella, pyoderma and dermatophytosis
Coat brushing, unstained adhesive tape strip, skin scrapings, trichogram, cytology, Wood's lamp examination, fungal culture, antibacterial therapy
No diagnosis
3. Perform focused diagnostic tests to differentiate remaining possibilities based on index of suspicion
Biopsy
Endocrine disease
Leishmaniasis
FeLV
FIV
Pemphigus
Lupus
Drug eruption
Cutaneous lymphoma
Hepatocutaneous syndrome
Primary scaling disorders
Haematology
Biochemistry
Endocrine disease
Liver/kidney failure
Leishmaniasis
Hepatocutaneous syndrome
Endocrine tests
Hypothyroidism
Hyperadrenocorticism
Serology for infectious agents
Leishmaniasis
FeLV
FIV
GI function tests
Malabsorption

Figure 6.1 General diagnostic approach to scaling disorders

7

Macular, papular and pustular diseases

Differential diagnosis

Macules are circular, flat areas of abnormally coloured skin, up to 1 cm in diameter. As described in Chapter 2, macules may occur due to erythema, haemorrhage or changes in skin pigmentation (hyperpigmented or hypopigmented macules). This chapter is concerned with erythematous and haemorrhagic macules. Macules associated with abnormal pigmentation will be discussed in Chapter 10.

Papules and pustules are small red spots. Pustules contain visible pus whereas papules do not. Papules and pustules often become crusted because exudate is deposited on the skin surface and becomes enmeshed within the stratum corneum. Papules and pustules may also result in circumferentially spreading circular lesions surrounded by a rim of scale (epidermal collarettes).

In some diseases such as staphylococcal pyoderma, macules, papules and pustules can occur together on the same animal because they represent a pathological continuum. In other diseases, one type of lesion may predominate. In either case, the animal has visible skin lesions that will appear as a rash. It is important that the clinician assesses these rashes carefully to identify the precise identity of the lesions because this has diagnostic significance. Tables 7.1, 7.2 and 7.3 show the differential diagnoses for macular, papular and pustular eruptions.

Clinical features of macular, papular and pustular eruptions

Some of the most common causes of these eruptions are pruritic. Hence, the clinical features of flea bite hypersensitivity, fly bite hypersensitivity, *Sarcoptes scabiei* infestation, staphylococcal pyoderma, contact dermatitis and miliary dermatitis are all described in Chapter 4. Demodicosis is described in Chapter 5 because this usually presents with alopecia. However, demodicosis may lead to secondary infection of the hair follicles and thus cause pustules.

The remainder of this chapter will describe some uncommon causes of macular, papular and pustular eruptions that need to be differentiated from the more common diseases listed above.

Erythema multiforme minor

Aetiology: A sudden onset, immune-mediated, erythematous eruption that can be associated with adverse reactions to drugs or viral infections. Erythema multiforme can also lead to ulcerative skin lesions (described in Chapter 8).

Predominant lesions:

Erythematous macules.

Table 7.1 Diseases that may lead to macular eruptions

Lesion type	Specific disease
Erythematous macules	**Staphylococcal pyoderma (D)** **Flea bite hypersensitivity (D and C)** Fly bite hypersensitivity (D and C) Contact dermatitis (D and C) Erythema multiforme minor (D and C)
Haemorrhagic macules	Vasculitis (D and C) Coagulopathies (D and C)

Those in bold would be most common in general practice. D = those diseases seen in dogs, C = those diseases seen in cats.

Table 7.2 Diseases that may lead to papular eruptions

Papules	**Staphylococcal pyoderma (D)** **Flea bite hypersensitivity (D and C)** Fly bite hypersensitivity (D and C) ***Sarcoptes scabiei* infestation (D)** **Atopic dermatitis (D and C)** Contact dermatitis (D and C) Feline cowpox infection (C) Miliary dermatitis (C)

Those in bold would be most common in general practice. D = those diseases seen in dogs, C = those diseases seen in cats.

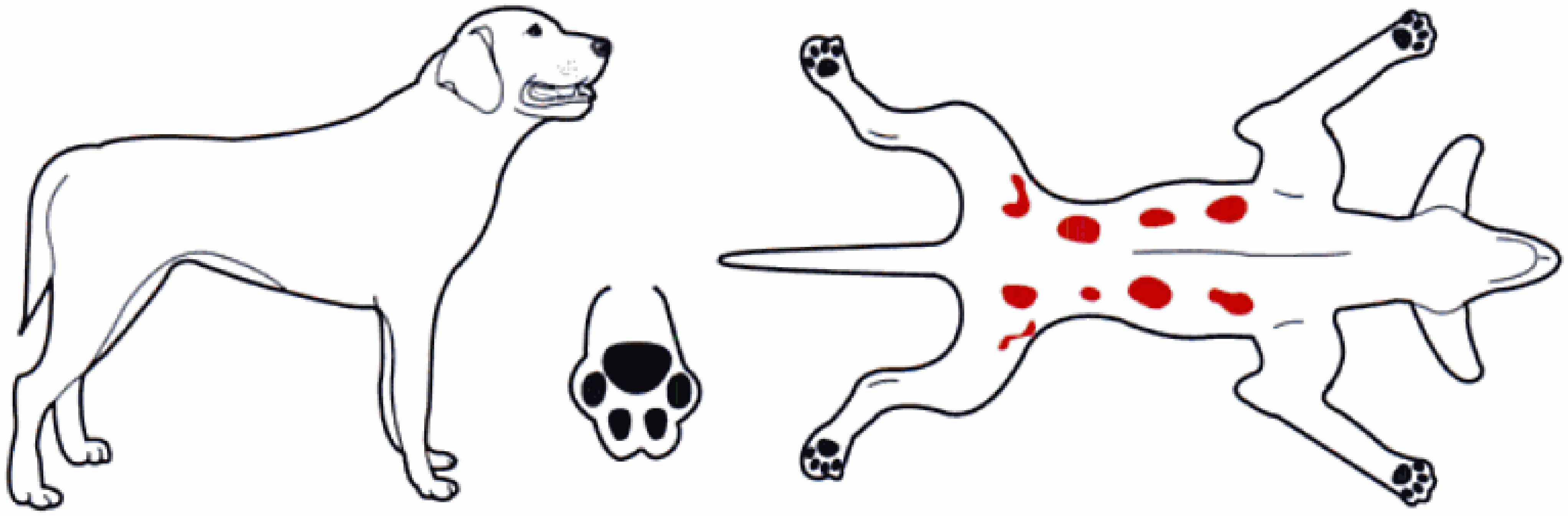
Table 7.3 Diseases that may lead to pustular eruptions

Pustules	**Staphylococcal pyoderma (D)** **Demodicosis (D)** Pemphigus foliaceus (D and C) Pemphigus erythematosus (D and C) Bullous impetigo (D) Sub-corneal pustular dermatosis (D) Sterile eosinophilic pustulosis (D)

Those in bold would be most common in general practice. D = those diseases seen in dogs, C = those diseases seen in cats.

Lesion distribution:

Most obvious on the ventrum but can be generalised.



Appropriate diagnostic tests:

Diascopy (pressing a glass slide onto the skin to see if it blanches)
Biopsy

Vasculitis

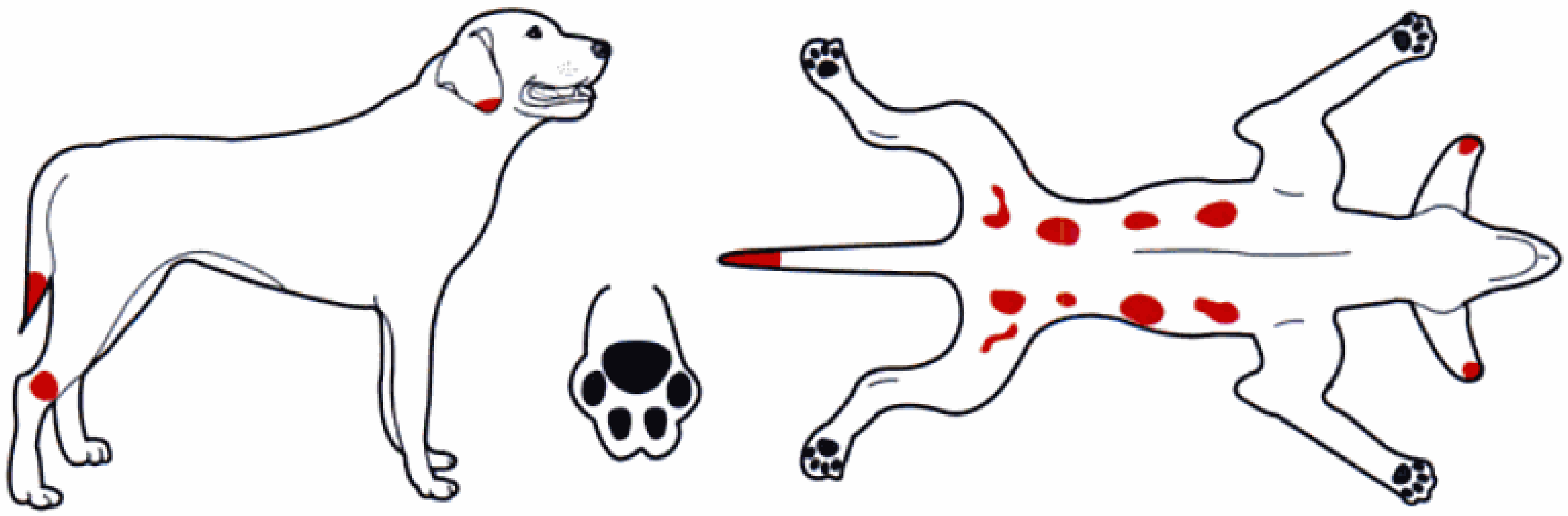
Aetiology: A sudden onset eruption caused by bleeding into the skin from inflamed blood vessels. May be associated with infections, systemic diseases, adverse reactions to drugs, lupus, or idiopathic. Vasculitis can also lead to ulcerative skin lesions (described in Chapter 8).

Predominant lesions:

Haemorrhagic macules (ecchymoses), purpura.

Lesion distribution:

Most obvious on the ventrum, extremities and over pressure points.



Appropriate diagnostic tests:

Diascopy (pressing a glass slide onto the skin to see if it blanches)
Biopsy

Pemphigus foliaceus

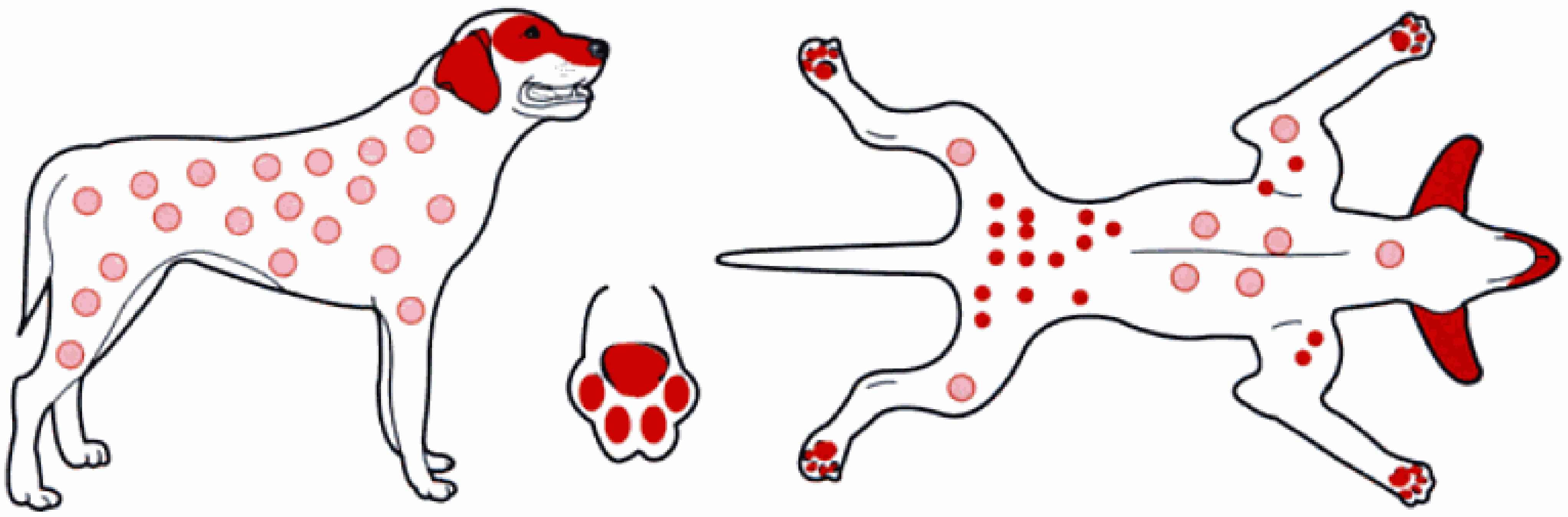
Aetiology: An autoimmune disease in which autoantibodies attack intercellular adhesion molecules in the epidermis leading to acantholysis (separation of the cells in the stratum spinosum).

Predominant lesions:

The primary lesions are pustules, but these rapidly become crusts, epidermal collarettes, alopecia, erosions.

Lesion distribution:

Usually starts on the face, nasal planum, pinnae. May also affect the footpads and become generalised.



Appropriate diagnostic tests:

Cytology
Biopsy

Pemphigus erythematosus

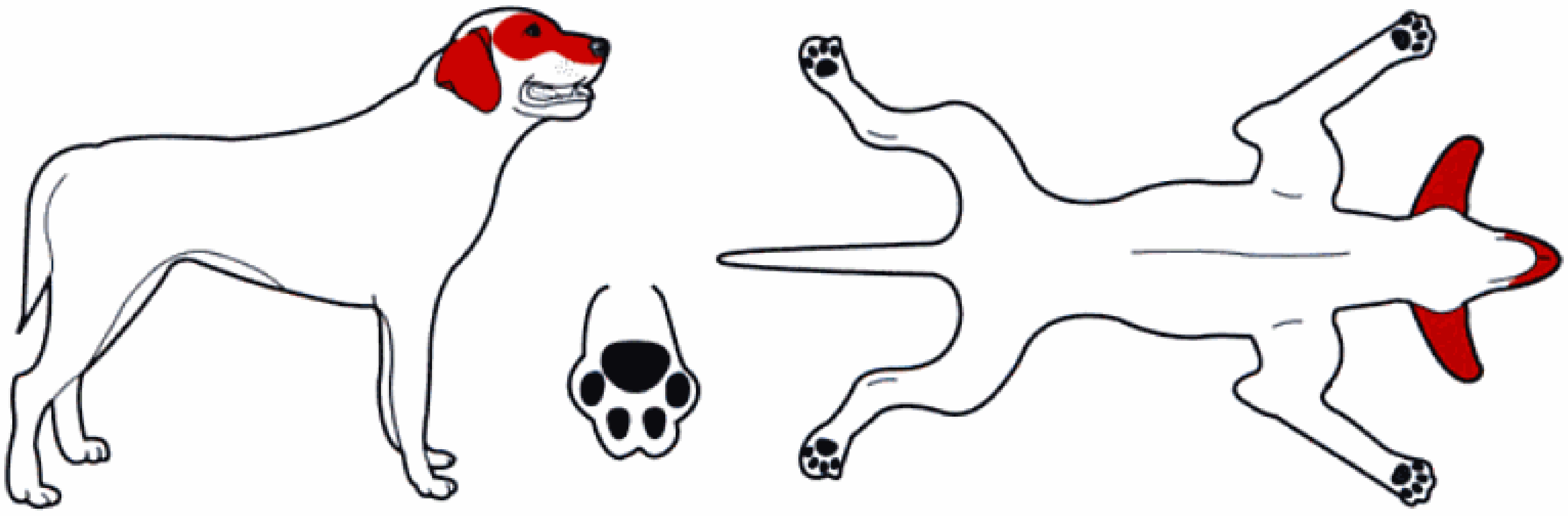
Aetiology: May be a more benign form of pemphigus foliaceus.

Predominant lesions:

Pustules, crusts, epidermal collarettes, alopecia, erosions.

Lesion distribution:

Face, nasal planum, and pinnae.



Appropriate diagnostic tests:

Cytology
Biopsy

Bullous impetigo

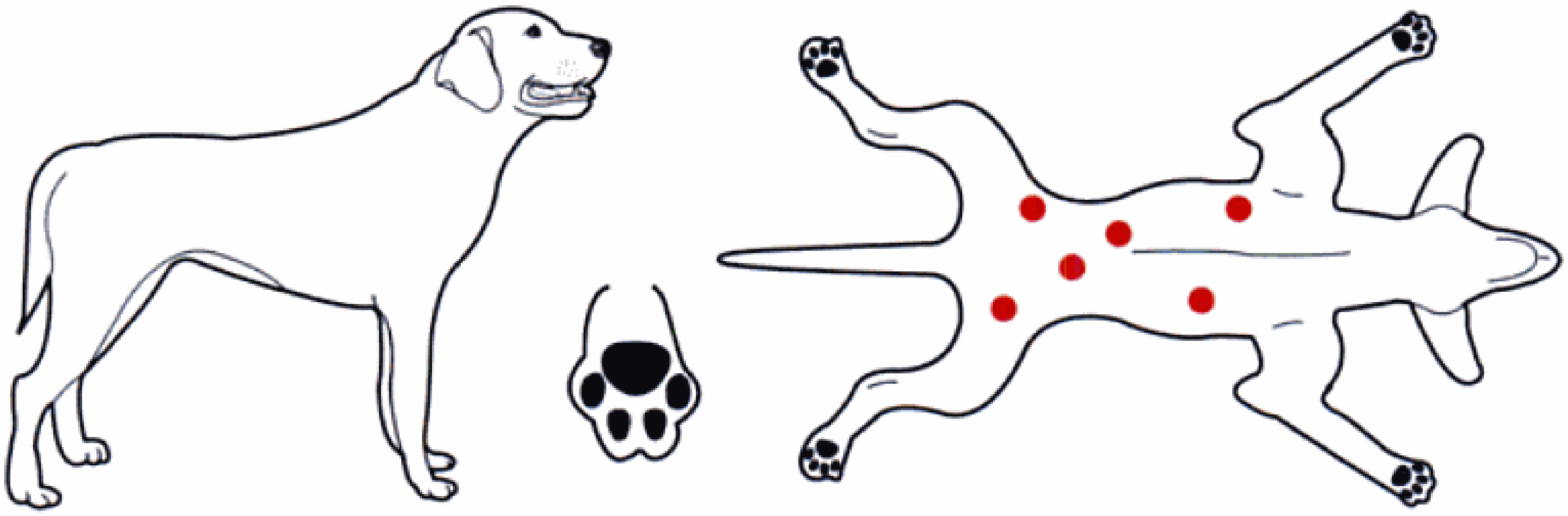
Aetiology: A staphylococcal skin infection usually secondary to systemic immunosuppressive diseases (most commonly hyperadrenocorticism) or immunosuppressive drugs.

Predominant lesions:

Large, flaccid pustules.

Lesion distribution:

Usually on the ventrum.



Appropriate diagnostic tests:

Cytology
Biopsy

Subcorneal pustular dermatosis

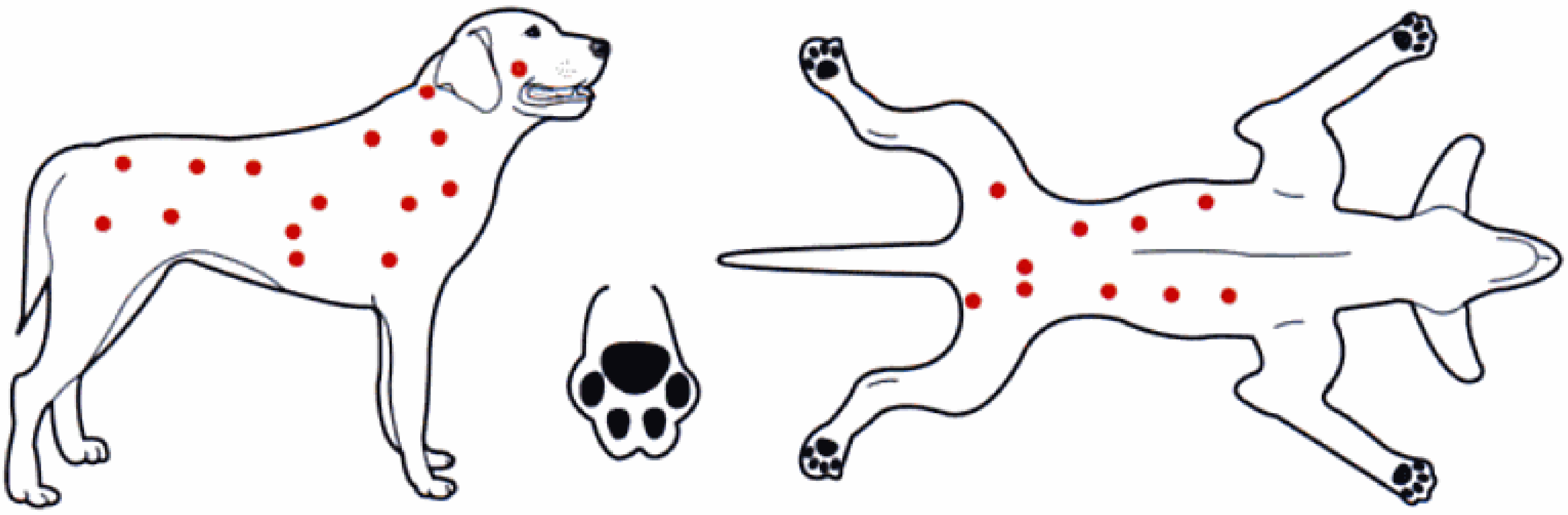
Aetiology: A very rare, idiopathic skin disease.

Predominant lesions:

Pustules, crusts, alopecia, epidermal collarettes, target lesions.

Lesion distribution:

Head and trunk.



Appropriate diagnostic tests:

Cytology
Biopsy

Sterile eosinophilic pustulosis

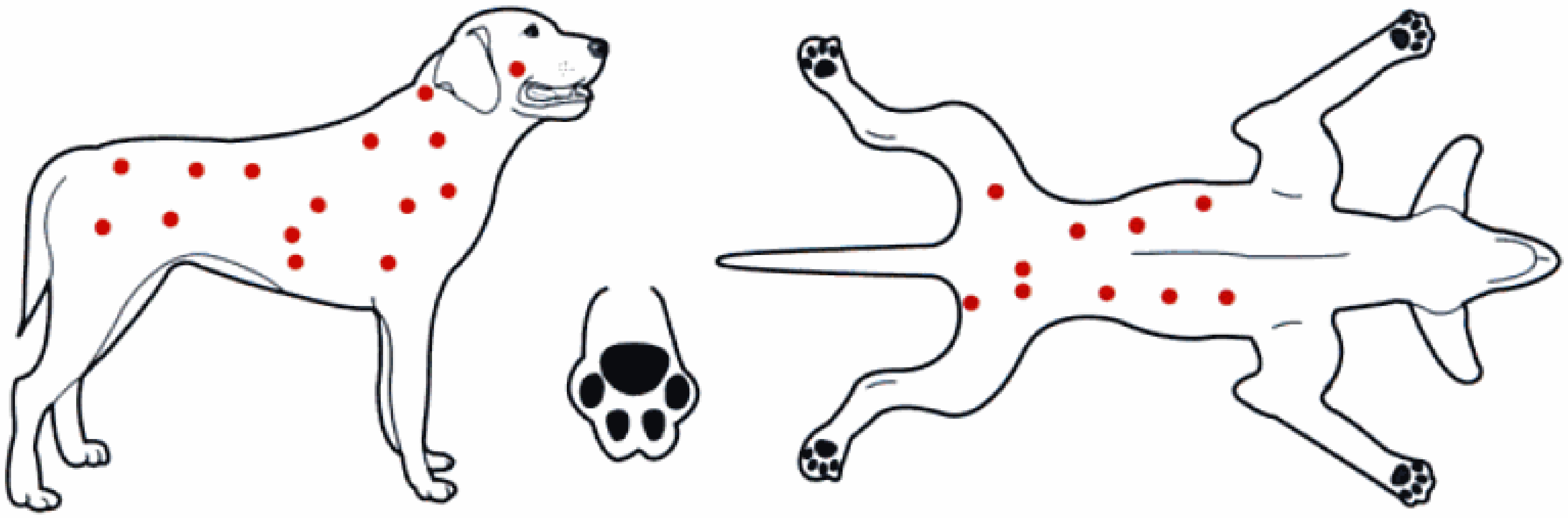
Aetiology: A very rare, idiopathic skin disease.

Predominant lesions:

Pustules, crusts, alopecia, epidermal collarettes, target lesions.

Lesion distribution:

Mainly on the trunk.



Appropriate diagnostic tests:

Cytology, Biopsy.

Feline cowpox infection

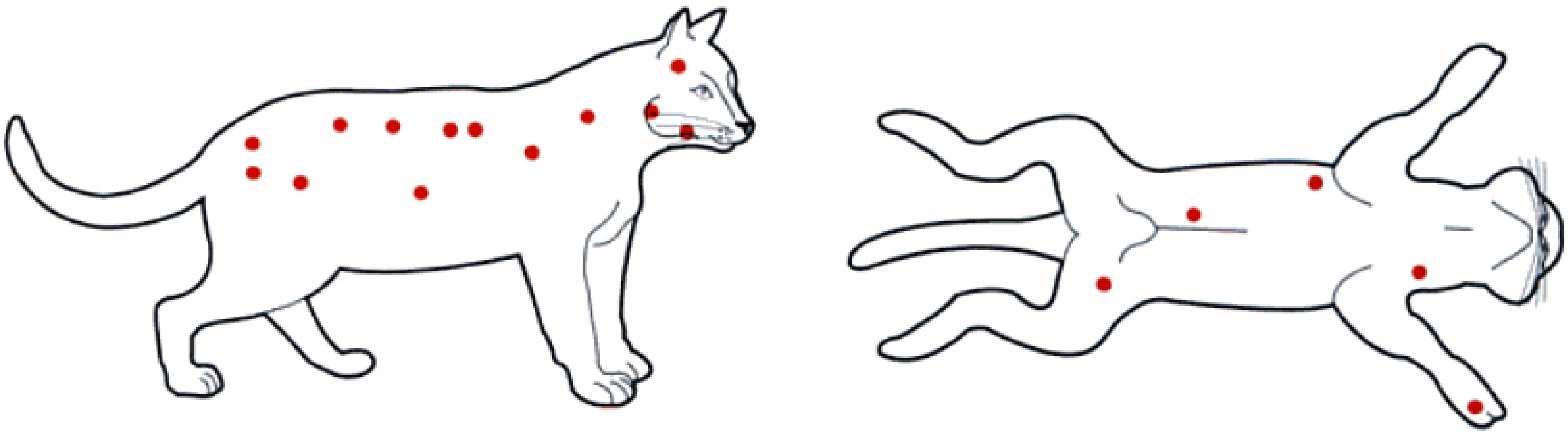
Aetiology: Infection of the skin with cowpox virus. Cowpox is restricted to Western Europe.

Predominant lesions:

Macules, papules, crusts.

Lesion distribution:

Mainly on the head and trunk.



Appropriate diagnostic tests:

Biopsy, Serology, Virus isolation, Electron microscopy.

General diagnostic approach to macular, papular and pustular diseases

When presented with these types of cutaneous eruptions, it is critical that the clinician carefully assesses the nature and type of skin lesions. Often, the animal may appear to be covered in crusts or scaly patches of alopecia and it is easy to be side-tracked into considering other differential diagnoses. The clinician must look extremely carefully for the primary lesions and use them for further investigation.

As with other cutaneous problems, the history and clinical appearance may be suggestive of a particular disease. For experienced clinicians or veterinary dermatologists, this may be possible even with quite rare diseases such as pemphigus foliaceus. However, if the clinician is unsure as to the most likely diagnosis, a logical diagnostic approach is required so that possible causes are not missed.

The clinician should first determine if the lesions are purely macular, papular, pustular, or if a mixture of lesions is present. Figures 7.1, 7.2 and 7.3 provide diagnostic algorithms for each of these types of lesion.

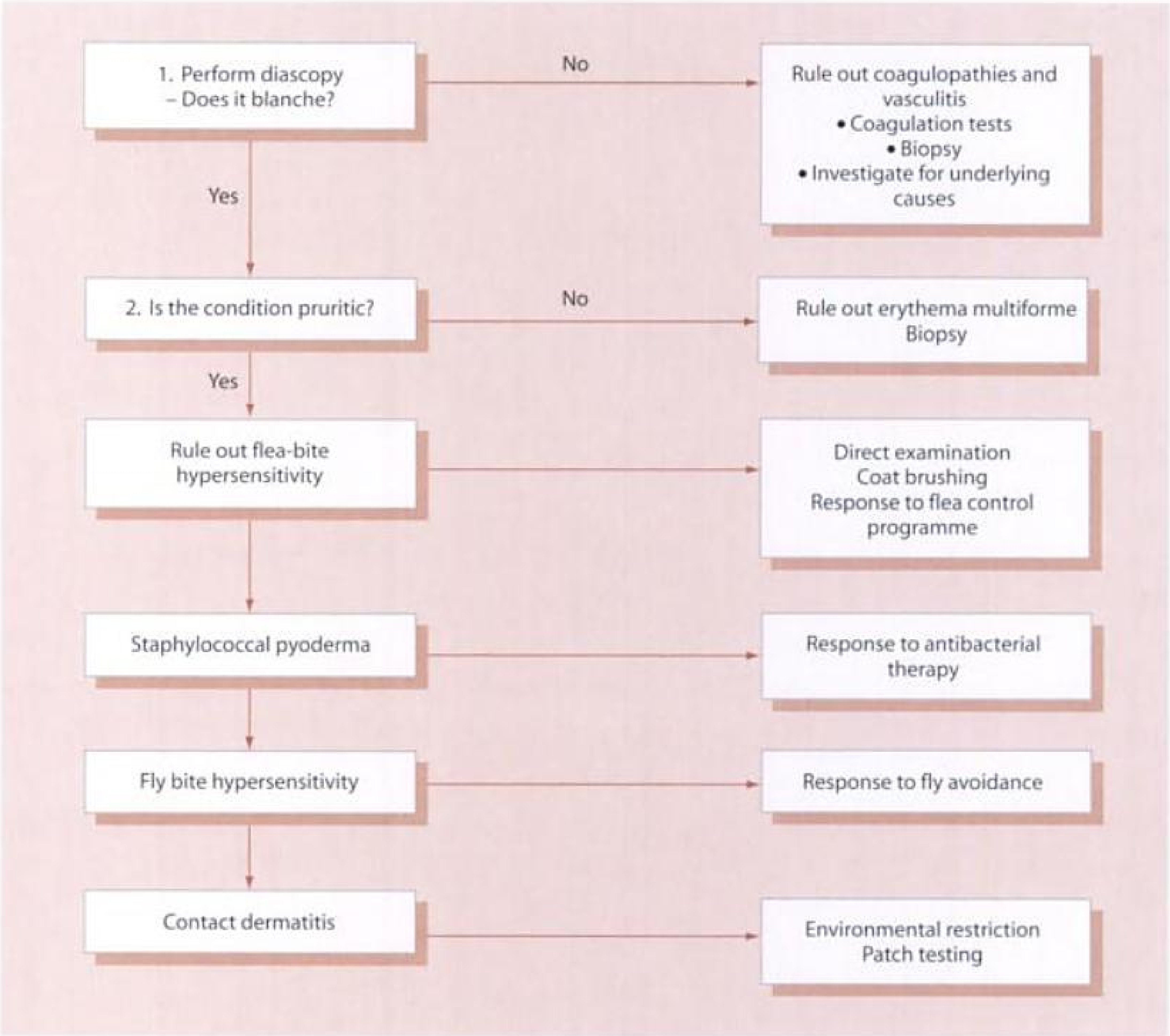

1. Perform diascopy
– Does it blanche?
No
Rule out coagulopathies and vasculitis
• Coagulation tests
• Biopsy
• Investigate for underlying causes
Yes
2. Is the condition pruritic?
No
Rule out erythema multiforme
Biopsy
Yes
Rule out flea-bite hypersensitivity
Direct examination
Coat brushing
Response to flea control programme
Staphylococcal pyoderma
Response to antibacterial therapy
Fly bite hypersensitivity
Response to fly avoidance
Contact dermatitis
Environmental restriction
Patch testing

Figure 7.1 General diagnostic approach to macular eruptions.

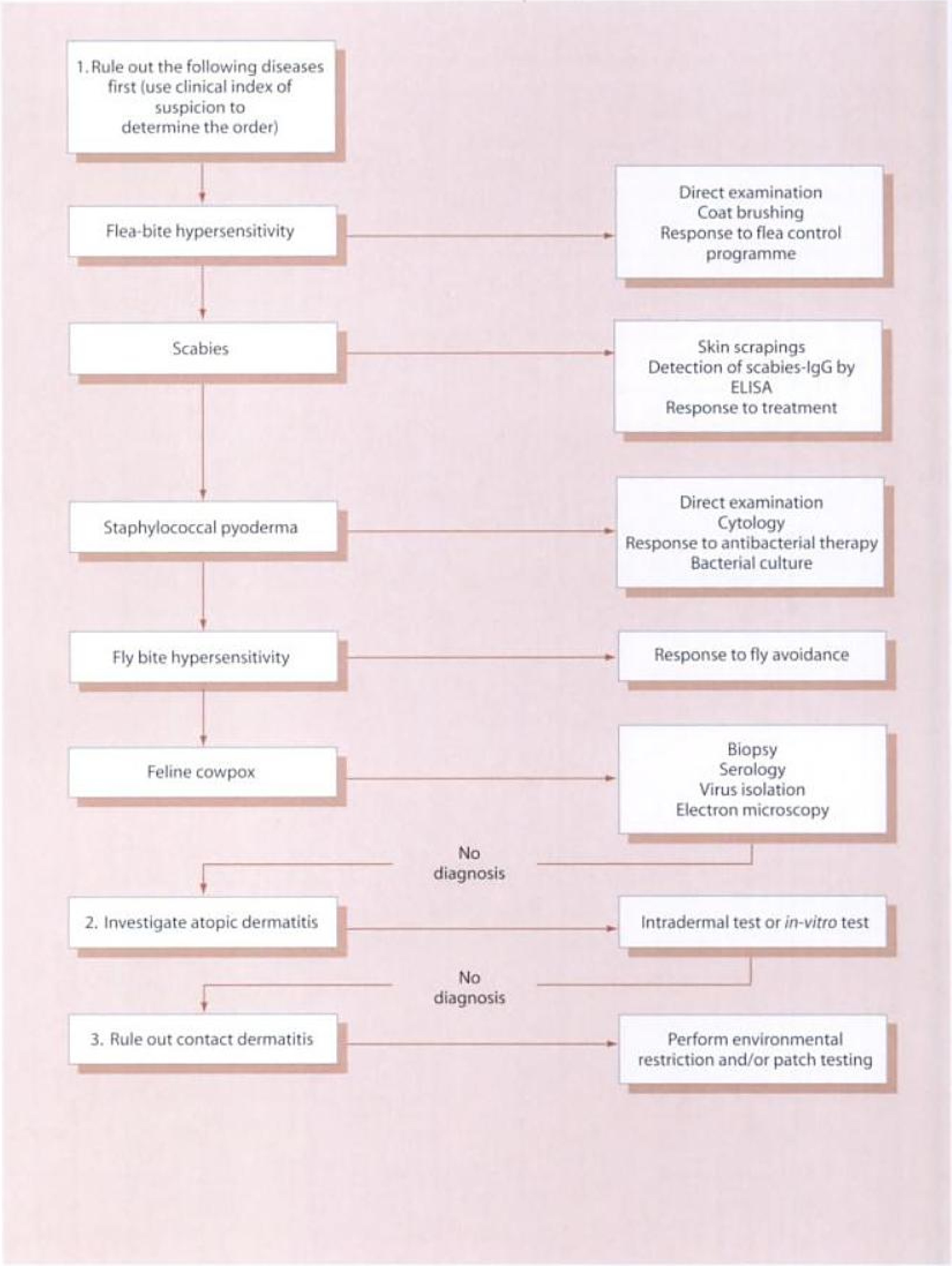

1. Rule out the following diseases first (use clinical index of suspicion to determine the order)
Flea-bite hypersensitivity
Direct examination
Coat brushing
Response to flea control programme
Scabies
Skin scrapings
Detection of scabies-IgG by ELISA
Response to treatment
Staphylococcal pyoderma
Direct examination
Cytology
Response to antibacterial therapy
Bacterial culture
Fly bite hypersensitivity
Response to fly avoidance
Feline cowpox
Biopsy
Serology
Virus isolation
Electron microscopy
No diagnosis
2. Investigate atopic dermatitis
Intradermal test or *in-vitro* test
No diagnosis
3. Rule out contact dermatitis
Perform environmental restriction and/or patch testing

Figure 7.2 General diagnostic approach to papular eruptions.

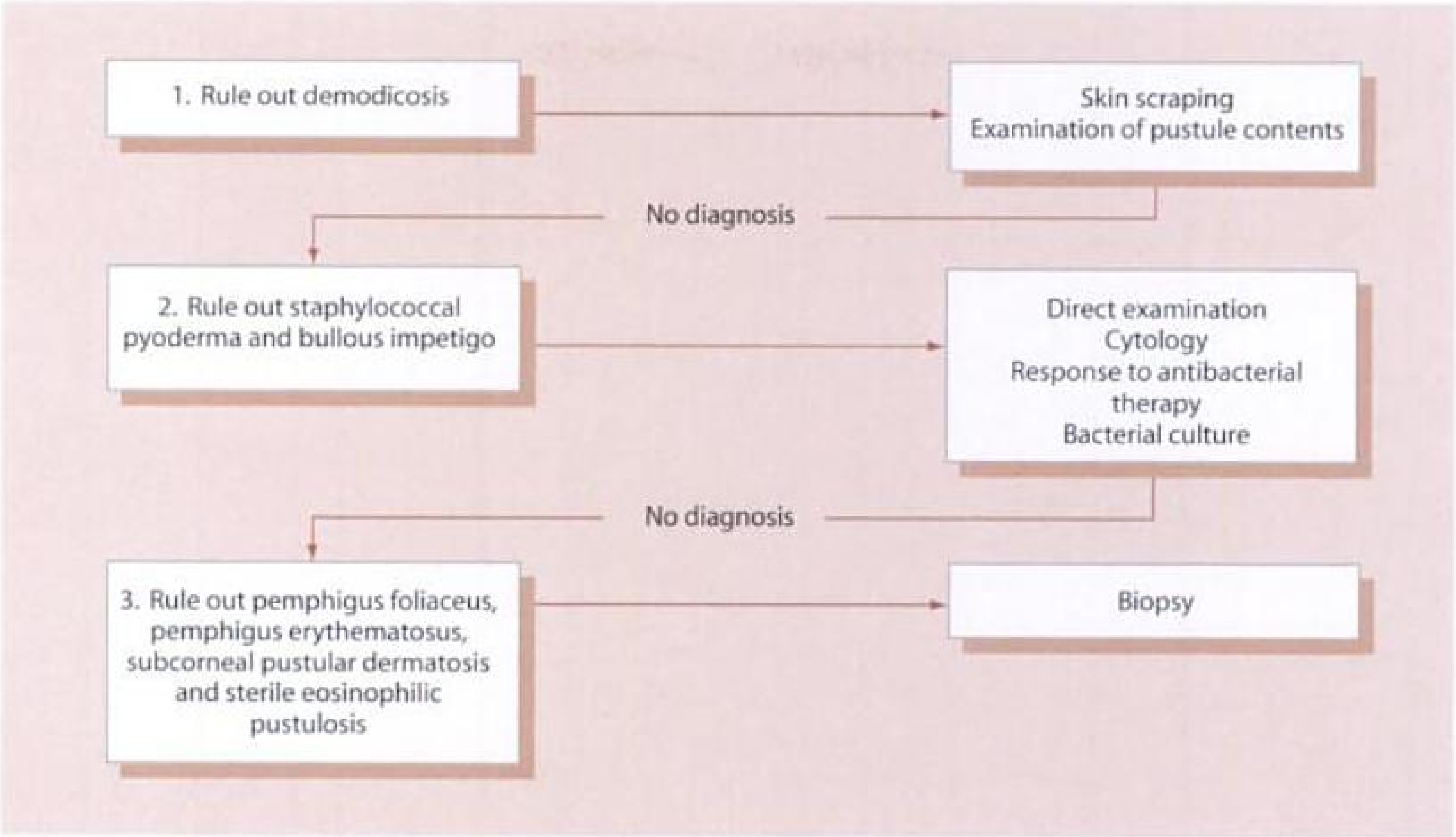

1. Rule out demodicosis
Skin scraping
Examination of pustule contents
No diagnosis
2. Rule out staphylococcal pyoderma and bullous impetigo
Direct examination
Cytology
Response to antibacterial therapy
Bacterial culture
No diagnosis
3. Rule out pemphigus foliaceus, pemphigus erythematosus, subcorneal pustular dermatosis and sterile eosinophilic pustulosis
Biopsy

Figure 7.3 General diagnostic approach to pustular eruptions.

8

Draining tracts and non-healing wounds

Differential diagnosis

Draining (sinus) tracts are openings between the underlying dermis or subcutis and the skin surface through which exudate is discharging. Draining tracts usually imply the presence of an infectious agent, either bacterial or fungal. In most cases, identification and elimination of the organism will lead to resolution of the skin disease. Draining tracts can also be seen in association with demodicosis (Chapter 5), but only when furunculosis and secondary infection have occurred. In some cases, draining tracts fail to respond to routine antimicrobial therapy. In this chapter, these persistent, non-responsive lesions will be described as non-healing wounds, although there may be no history of an actual traumatic wound at the onset of the condition. In rare cases, discharging sinuses can be sterile and result from immune-mediated diseases. Table 8.1 shows the major differential diagnoses for draining tracts in dogs and cats.

Table 8.1 Differential diagnoses for draining tracts and non-healing wounds

Aetiological category	Specific disease
Bacterial	**Abscesses (D and C)**
	Deep pyoderma (furunculosis) (D)
	Foreign bodies (D and C)
	Actinomycosis (D and C)
	Actinobacillosis (D and C)
	Nocardiosis (D and C)
	Mycobacterial infections (D and C)
Fungal	Kerion (D)
	Mycetoma and pseudomycetoma (D and C)
	Deep or systemic fungal infections (D and C)
Parasitic	**Demodicosis (D)**
Miscellaneous	Panniculitis (D and C)
	Juvenile cellulitis (D)
	Perianal fistulae (D)
	Focal metatarsal fistulae (D)
Congenital	Dermoid sinus (D)

Those in bold would be most common in general practice. D = those diseases seen in dogs, C = those diseases seen in cats.

Clinical features of skin diseases presenting with draining tracts

Abscesses

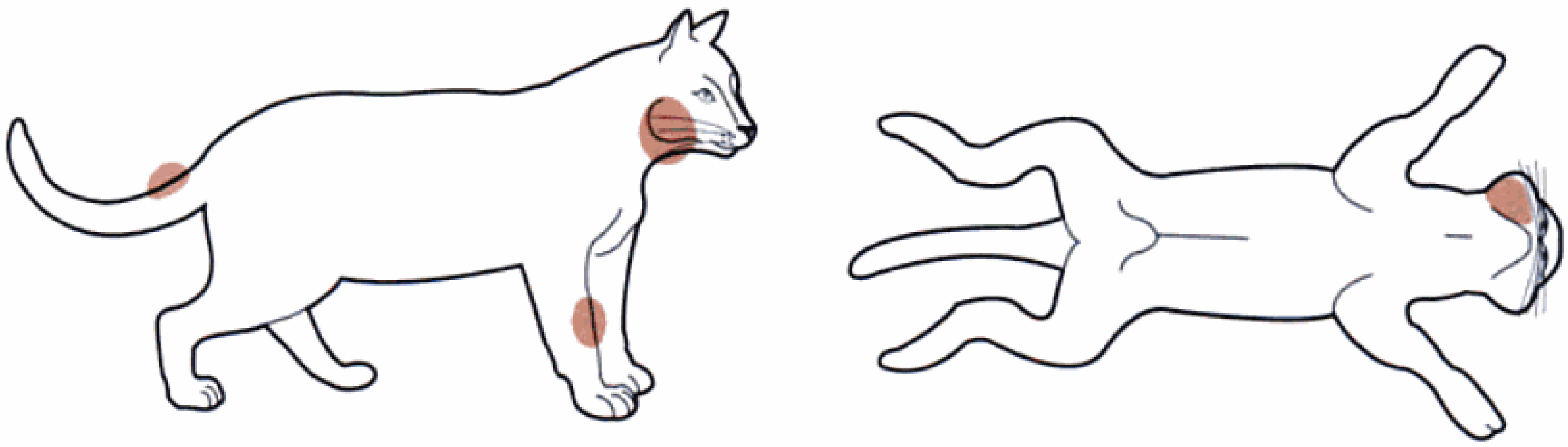
Aetiology: A subcutaneous accumulation of pus. Abscesses caused by *Pasteurella multocida* are extremely common in cats secondary to bite wounds from other cats. Abscesses are less common in dogs and may be caused by bite wounds, tooth root infections, infected anal sacs or foreign bodies.

Predominant lesions:

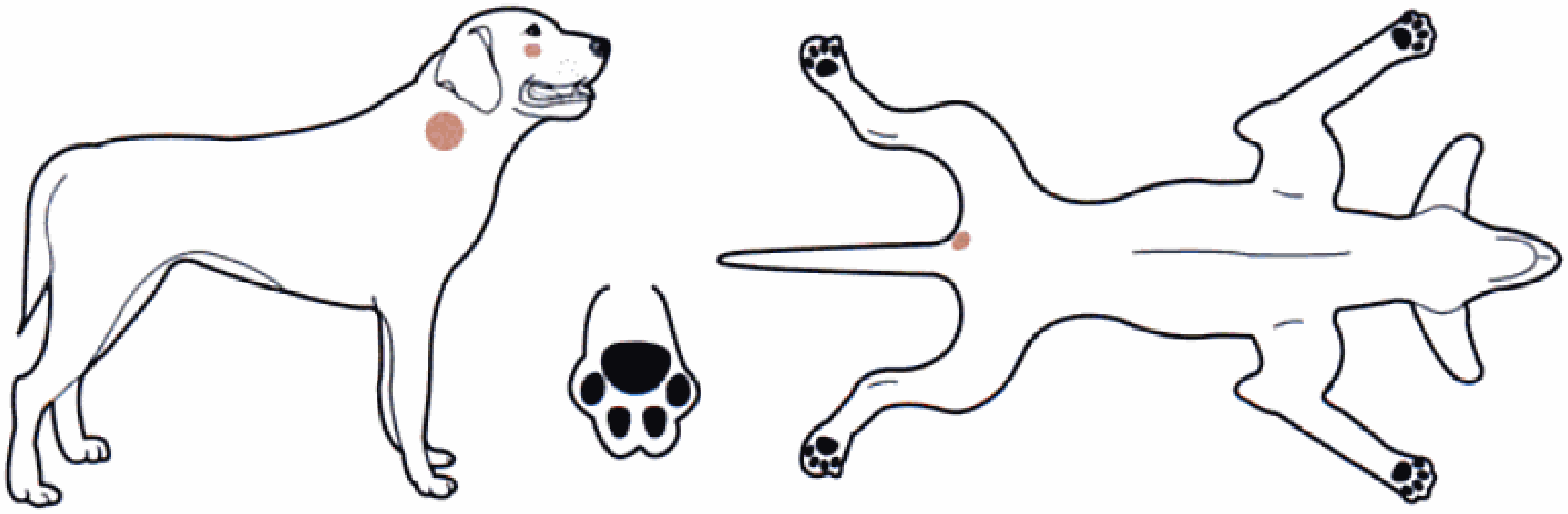
Subcutaneous swellings that may or may not lead to draining tracts.

Lesion distribution:

Cats – face, tail, legs, rest of body.



Dogs – site of bite wounds, below the eye (carnassial tooth root abscess), lateral to the anus (anal sac abscess), site of foreign bodies.



Appropriate diagnostic tests:

Clinical diagnosis
Lancing or fine needle aspiration
Cytology

Deep pyoderma

Aetiology: A deep bacterial infection of the dermis and/or subcutis usually caused by *Staphylococcus intermedius*, but occasionally associated with other bacteria such as *Pseudomonas* *aeruginosa*, *Proteus mirabilis* and *E. coli*. Deep pyodermas do not develop spontaneously and the clinician should always attempt to identify the underlying cause. With localised lesions, this may be bite wounds, foreign bodies or penetrating injuries. With widespread lesions, potential underlying causes include demodicosis, allergies, hypothyroidism, hyperadrenocorticism, internal disease, neoplasia, immunodeficiency and immunosuppressive drugs.

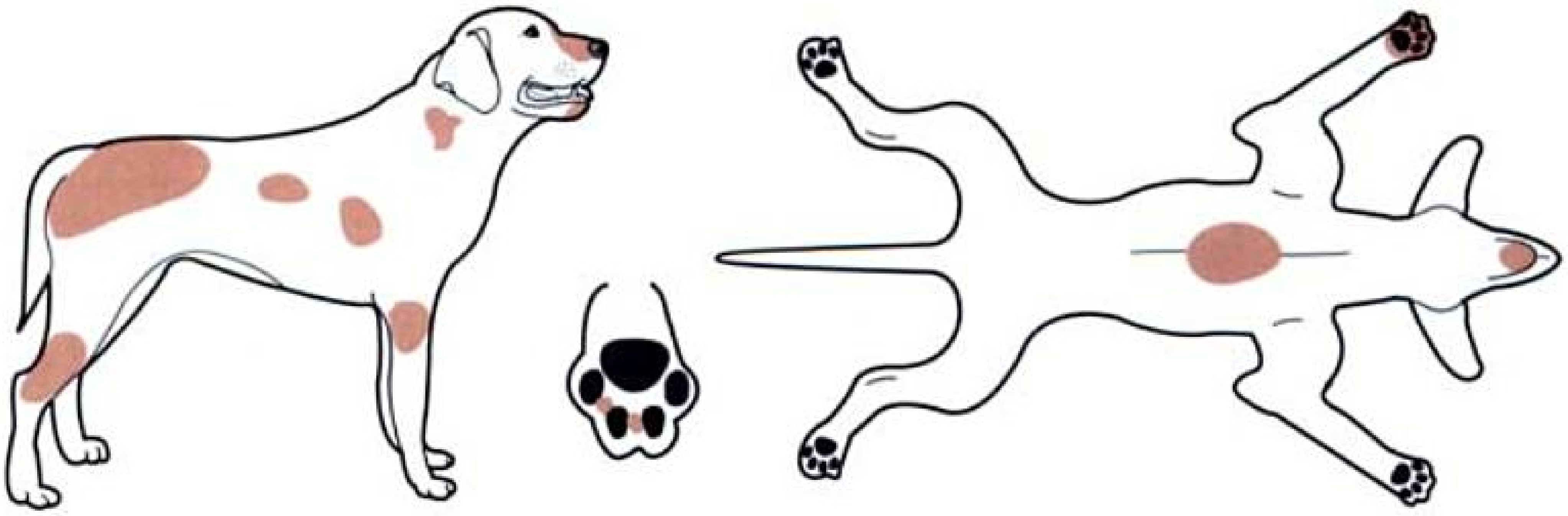
Predominant lesions:

Draining tracts, nodules.

Lesion distribution:

Various distribution patterns are recognised:

- Chin furunculosis (canine acne) – chin
- Nasal furunculosis – bridge of nose
- Interdigital furunculosis – interdigital skin
- Pressure point (callus) pyoderma – hocks, elbows, sternum
- Generalised furunculosis and cellulitis – trunk and legs (most common in German shepherd dogs and English bull terriers but can occur in other breeds).



Appropriate diagnostic tests:

Skin scraping
Cytology
Bacterial culture
Biopsy

Tests for potential underlying causes based on index of suspicion:

Haematology
Biochemistry
Thyroid function tests
Adrenal function tests
Allergy tests
Immune function tests
Radiography and ultrasound

Foreign bodies

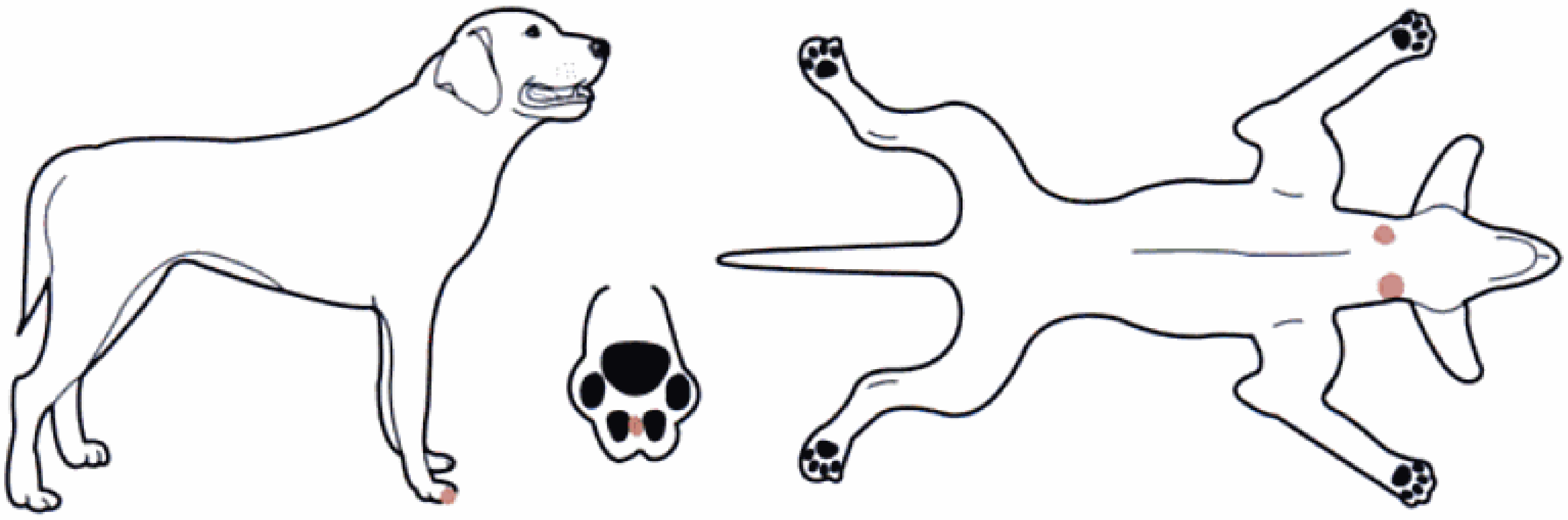
Aetiology: Subcutaneous infection caused by penetration of a foreign body such as a grass seed or a piece of wood.

Predominant lesions:

Discharging sinuses.

Lesion distribution:

Grass seeds – interdigital skin but can migrate to other sites.
Wood splinters – ventral neck following pharyngeal penetration.



Appropriate diagnostic tests:

Exploration of the sinus tract
Cytology

Actinomycosis (actinomycotic mycetoma)

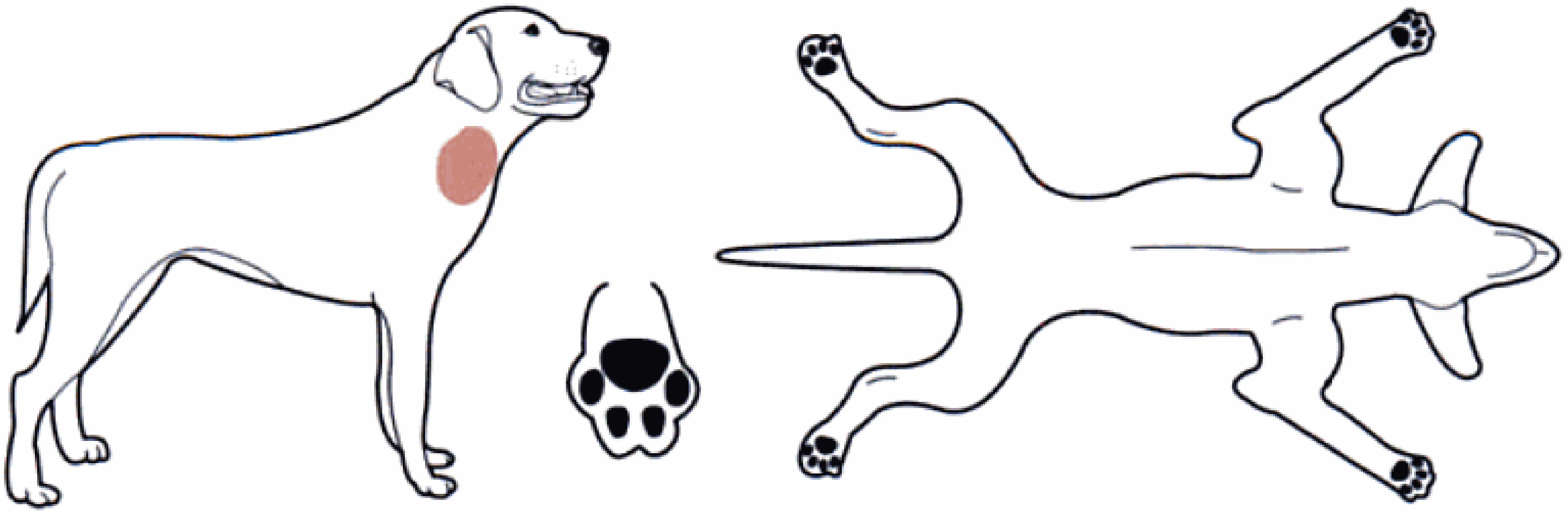
Aetiology: A rare subcutaneous infection caused by *Actinomyces* organisms which are usually introduced through penetrating wounds or foreign bodies

Predominant lesions:

Abscesses and draining tracts, non-healing wounds.

Lesion distribution:

At the site of the original wound.



Appropriate diagnostic tests:

Cytology
Bacterial culture
Biopsy

Actinobacillosis

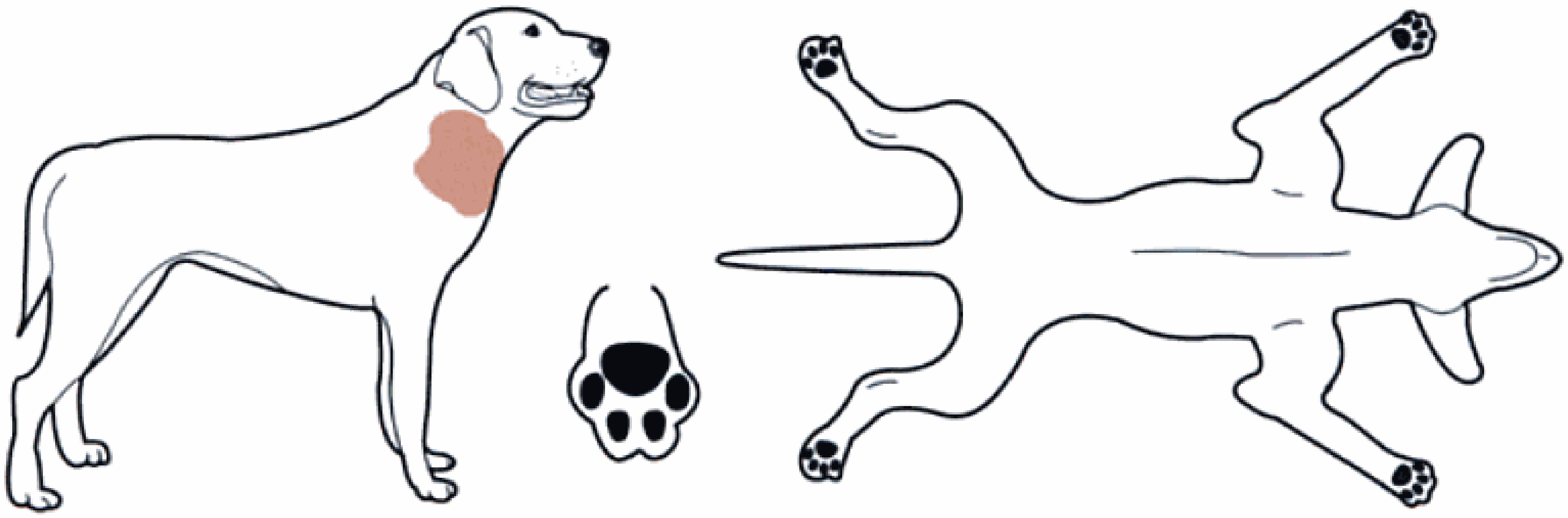
Aetiology: A rare subcutaneous infection caused by *Actinobacillus lignieresii* which is usually introduced through traumatic wounds.

Predominant lesions:

Abscesses and draining tracts, non-healing wounds.

Lesion distribution:

Often head, neck, mouth and limbs.



Appropriate diagnostic tests:

Cytology
Bacterial culture
Biopsy

Nocardiosis (actinomycotic mycetoma)

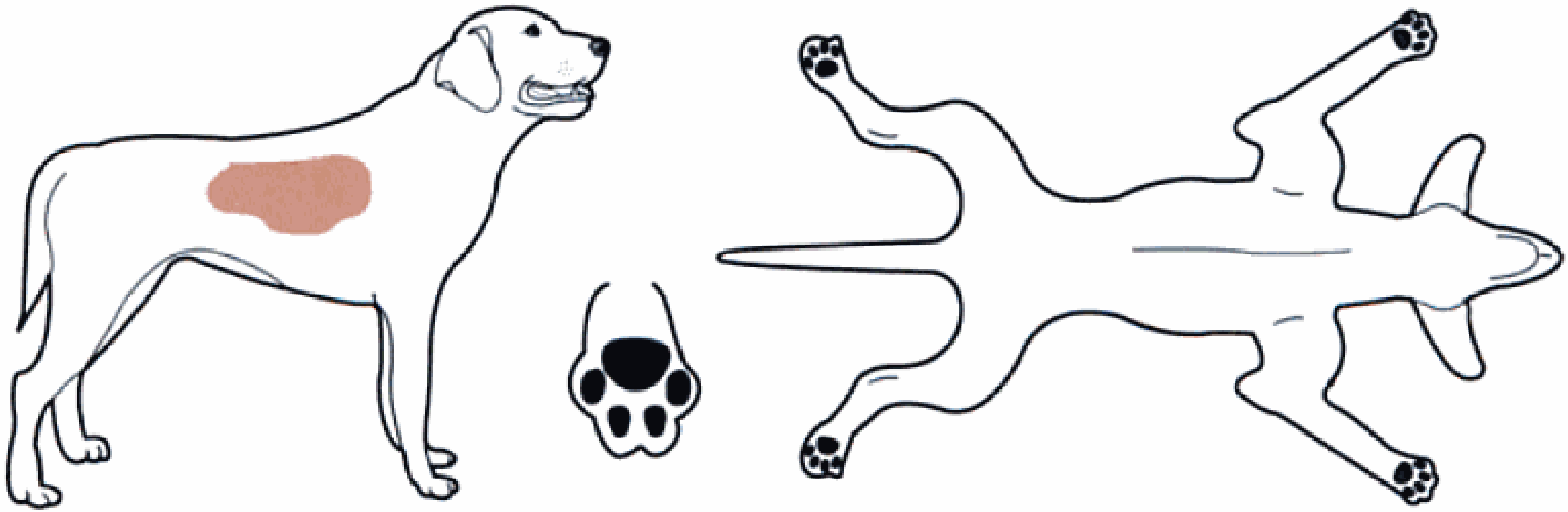
Aetiology: A rare subcutaneous infection caused by *Nocardia* organisms which are usually introduced through traumatic wounds.

Predominant lesions:

Abscesses and draining tracts, non-healing wounds.

Lesion distribution:

At the site of initial wounding.



Appropriate diagnostic tests:

Cytology
Bacterial culture
Biopsy

Mycobacterial infections

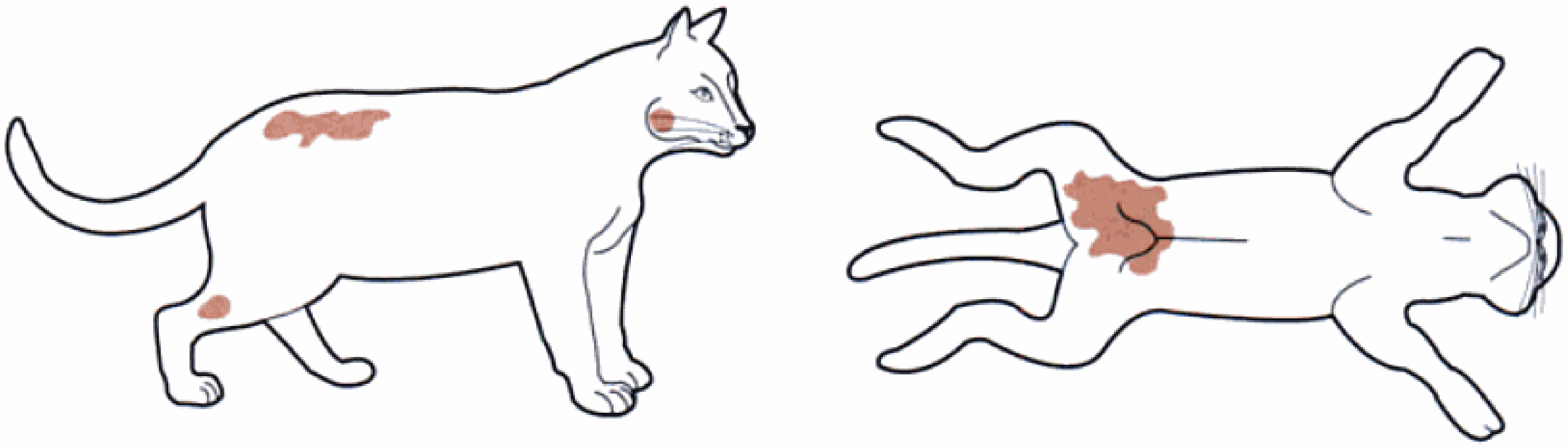
Aetiology: Rare cutaneous infections with various mycobacteria. *Mycobacterium tuberculosis, M. bovis, M. avium* and an unnamed variant cause tuberculosis; *M. lepraemurium* causes feline leprosy; and various atypical (fast growing) mycobacteria cause opportunistic infections. Mycobacterial infections are more common in cats than dogs.

Predominant lesions:

Ulcerated nodules, abscesses and draining tracts, non-healing wounds.

Lesion distribution:

Tuberculosis and feline leprosy – head, neck, limbs.
Atypical mycobacterial infections – inguinal region, lumbar region.



Appropriate diagnostic tests:

Cytology
Mycobacterial culture or PCR
Biopsy

Kerion

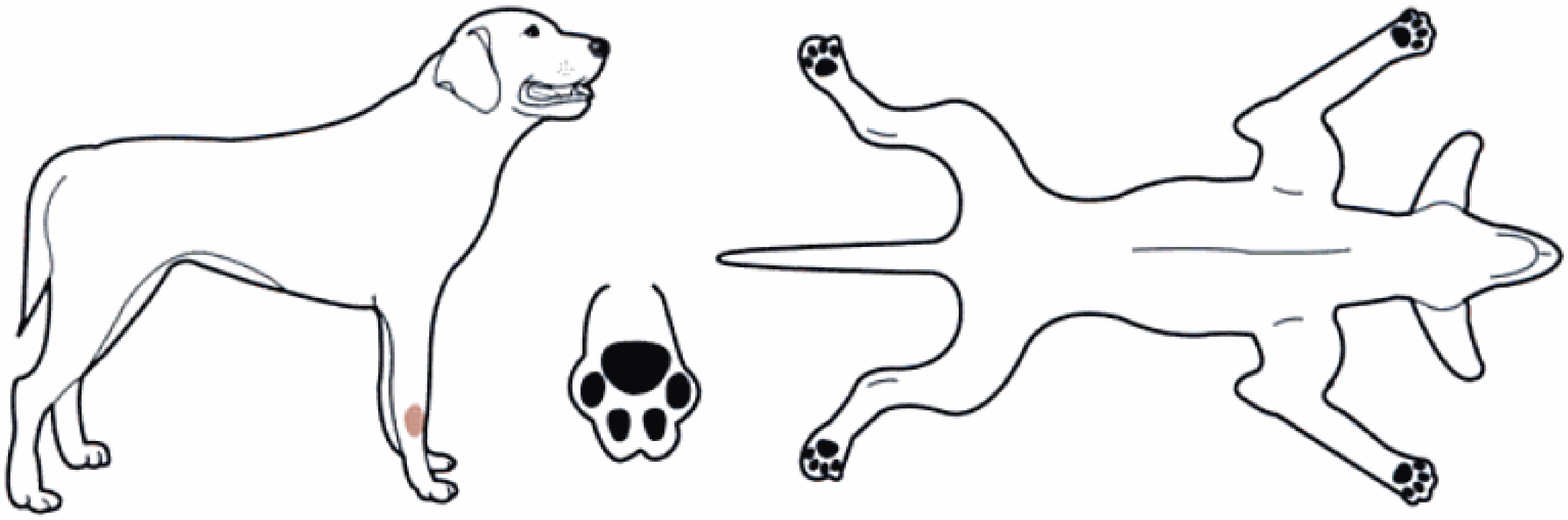
Aetiology: A deep infection with a dermatophyte, often *Microsporum gypseum* or *Trichophyton mentagrophytes.*

Predominant lesions:

Well circumscribed, boggy, nodular lesions with draining tracts.

Lesion distribution:

Face, distal limbs.



Appropriate diagnostic tests:

Cytology
Fungal culture
Biopsy

Mycetoma (eumycotic mycetoma) and pseudomycetoma

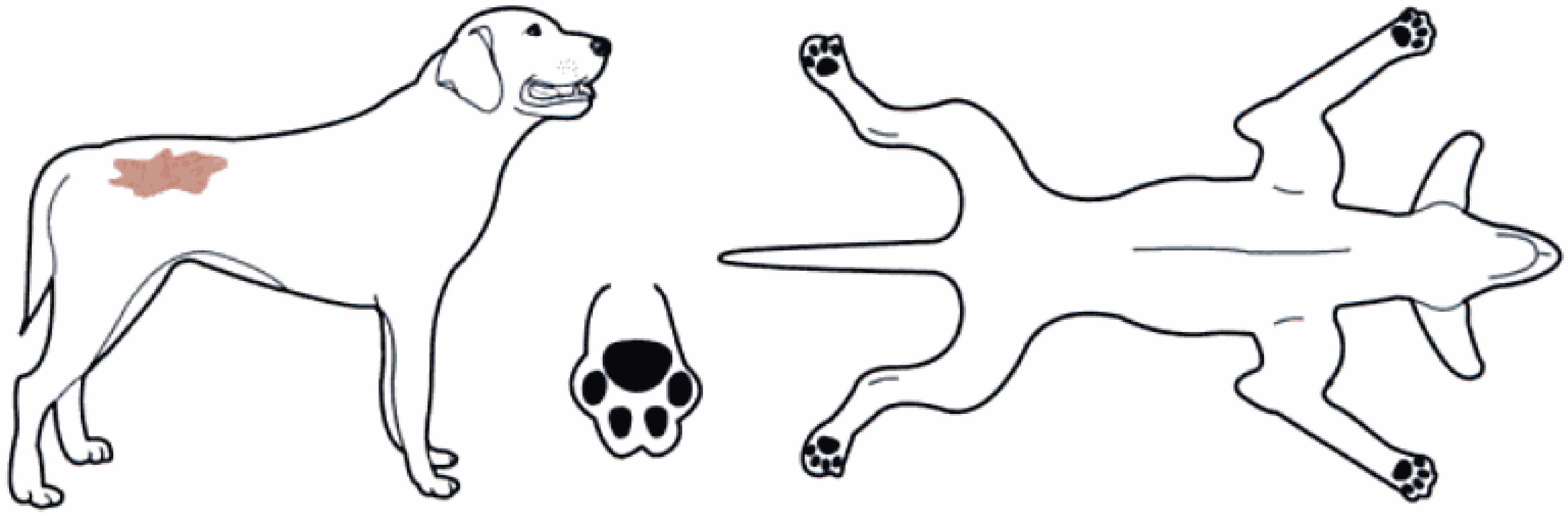
Aetiology: Subcutaneous infection with various saprophytic soil fungi (mycetoma) or dermatophytes (pseudomycetoma), usually following traumatic implantation.

Predominant lesions:

Swelling, draining tracts and pus containing sulphur granules (grains). Sulphur granules are grossly visible accumulations of mycelial hyphae.

Lesion distribution:

At the site of original wound.



Appropriate diagnostic tests:

Cytology
Fungal culture
Biopsy

Deep and systemic fungal infections (sporotrichosis, phaeohyphomycosis, pythiosis, blastomycosis, cryptococcosis, coccidiomycosis, histoplasmosis)

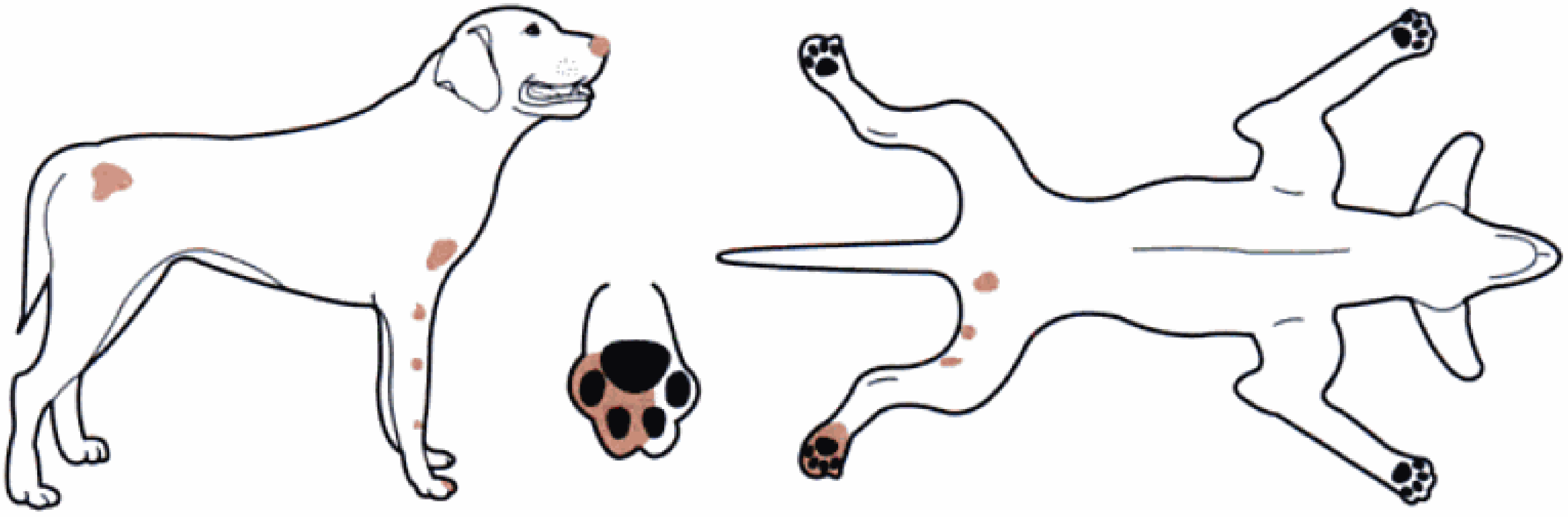
Aetiology: Subcutaneous infection with various fungi, some of which can cause systemic infections.

Predominant lesions:

Nodules, ulcers, draining tracts, non-healing wounds, lymphadenopathy
Systemic infections can cause anorexia, pyrexia, weight loss, coughing, dyspnoea, gastrointestinal disease, ocular disease, central nervous system disease.

Lesion distribution:

Head, nasal planum, limbs, clawbeds but can be anywhere.



Appropriate diagnostic tests:

Cytology
Fungal culture
Biopsy
Tests appropriate for the systemic signs

Panniculitis

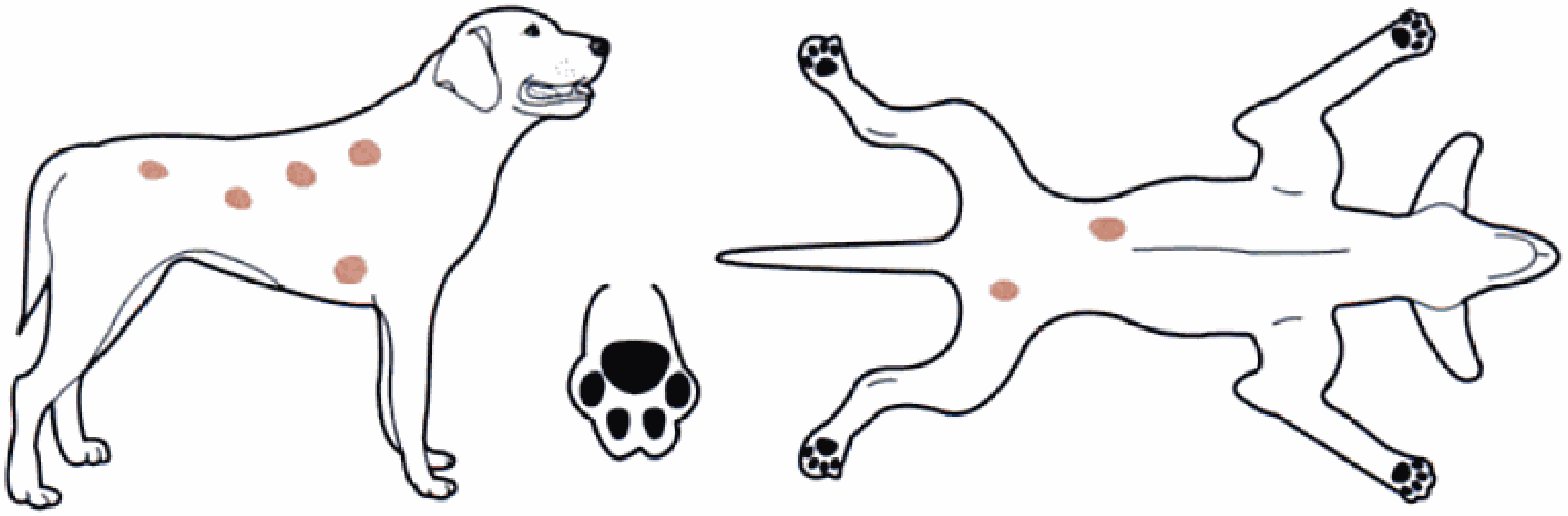
Aetiology: Inflammation of the subcutaneous fat. Panniculitis may be caused by infectious agents (most of the agents listed above), lupus, pancreatic disease, or vitamin E deficiency. An idiopathic form is also recognised (sterile nodular panniculitis).

Predominant lesions:

Nodules, draining tracts (exudate is usually oily).

Lesion distribution:

Usually over the trunk.



Appropriate diagnostic tests:

Cytology
Bacterial and fungal culture
Biopsy
Tests appropriate for the underlying causes

Juvenile cellulitis (sterile granulomatous dermatitis and lymphadenitis, big head)

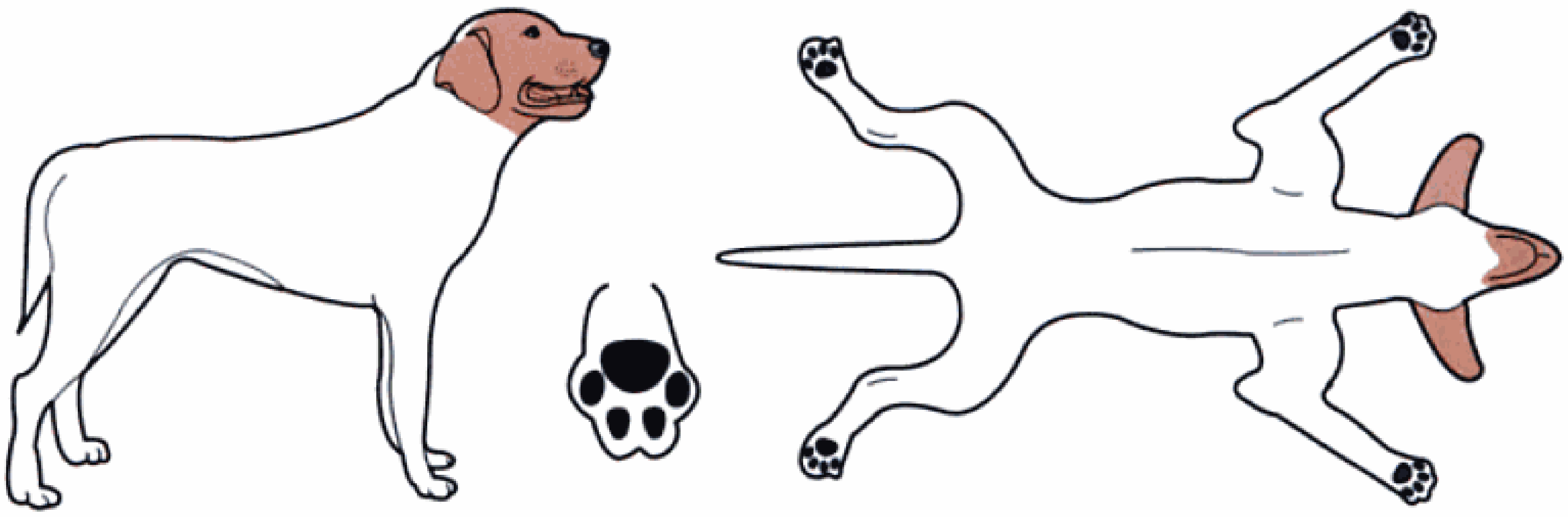
Aetiology: An idiopathic inflammatory disease of dogs that usually occurs between 3 weeks and 4 months of age.

Predominant lesions:

Acute skin swelling with draining tracts; purulent otitis externa; marked submandibular lymphadenopathy.

Lesion distribution:

Eyelids, lips, muzzle, nose, pinnae.



Appropriate diagnostic tests:

Skin scraping
Cytology

Focal metatarsal fistulation

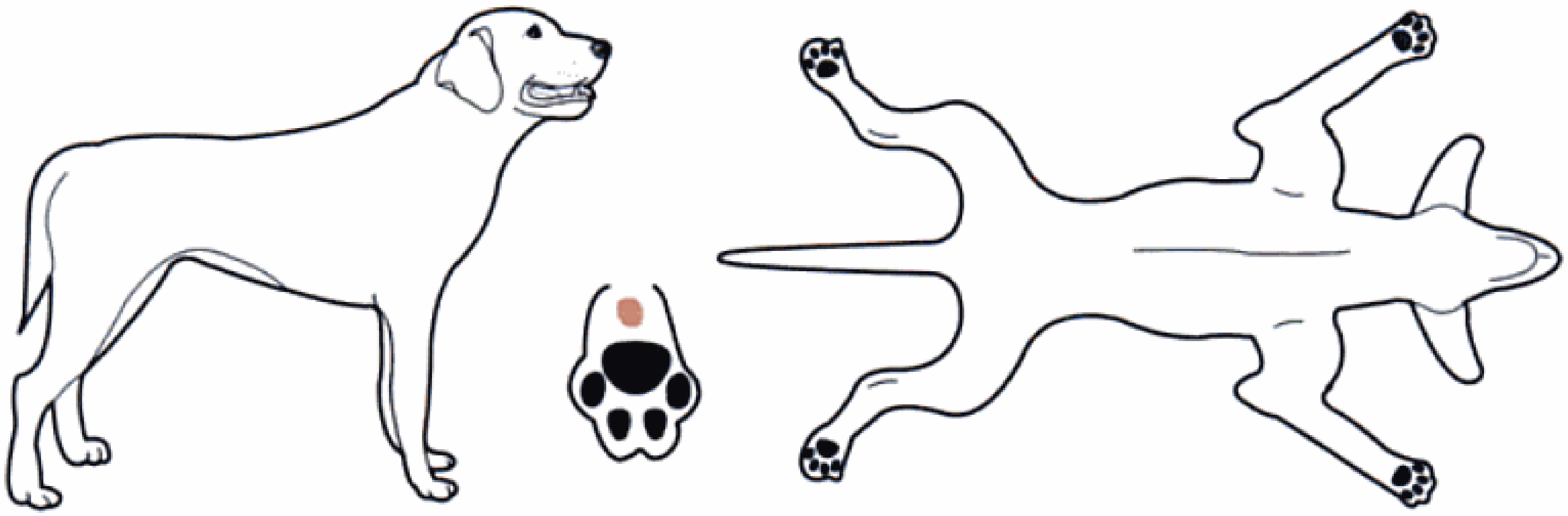
Aetiology: An uncommon, idiopathic disorder of German shepherd dogs.

Predominant lesions:

Solitary draining tracts.

Lesion distribution:

Ventral aspect of the metatarsus or metacarpus, just proximal to the metatarsal or metacarpal pad.



Appropriate diagnostic tests:

Cytology
Bacterial culture
Biopsy

Perianal fistulae

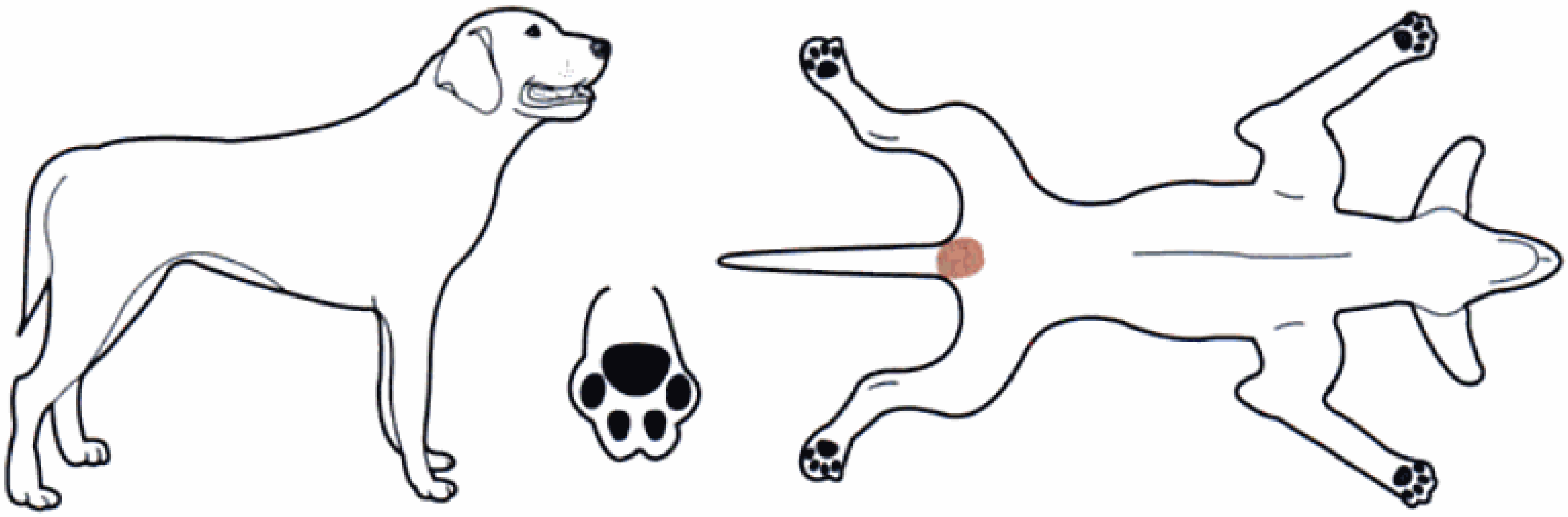
Aetiology: A disorder affecting mainly German shepherd dogs but occasionally seen in other breeds. May be immune-mediated.

Predominant lesions:

Ulcers, draining tracts.

Lesion distribution:

Around the anus.



Appropriate diagnostic tests:

Clinical diagnosis
Cytology
Bacterial culture

Dermoid sinus

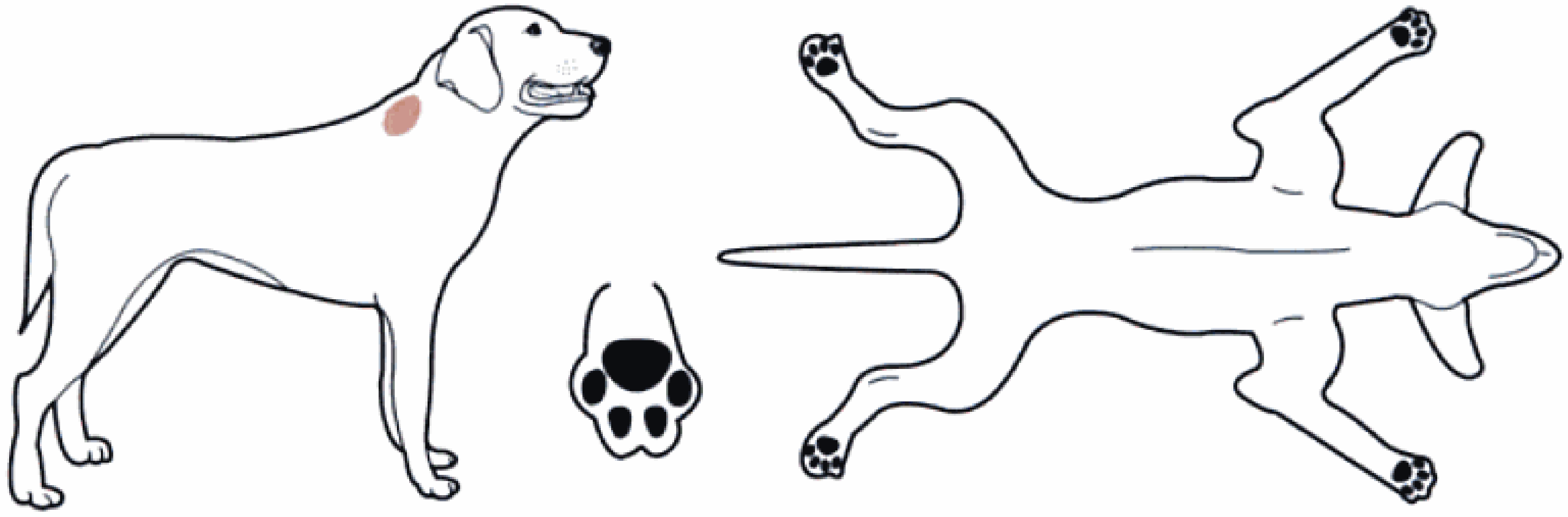
Aetiology: An uncommon, congenital disorder of Rhodesian Ridgebacks and occasionally other breeds. Caused by incomplete separation of the neural tube and skin during embryonic development.

Predominant lesions:

Solitary draining tracts.

Lesion distribution:

Dorsal neck.



Appropriate diagnostic tests:

Cytology
Bacterial culture
Fistulogram

General diagnostic approach to draining tracts and non-healing wounds

When confronted with an animal with draining tracts or non-healing wounds, the clinician should always be aware of the possibility of a zoonotic infection. Mycobacterial infections, sporotrichosis, and blastomycosis can lead to severe infections in people. As a precaution, clinicians should wear gloves when examining animals with draining tracts and be extra cautious when performing diagnostic tests with scalpel blades and needles.

The most important clinical objective when presented with draining tracts is to determine if an infectious agent is present, and if so, what type. Some conditions such as cat bite abscesses are so common that an initial diagnosis can be made clinically. In other cases, diagnostic tests are mandatory before a diagnosis can be made. These cases may require specialised culture techniques to identify some of the infectious agents described in this chapter.

The basic diagnostic approach to draining tracts and non-healing wounds is described in Figure 8.1.

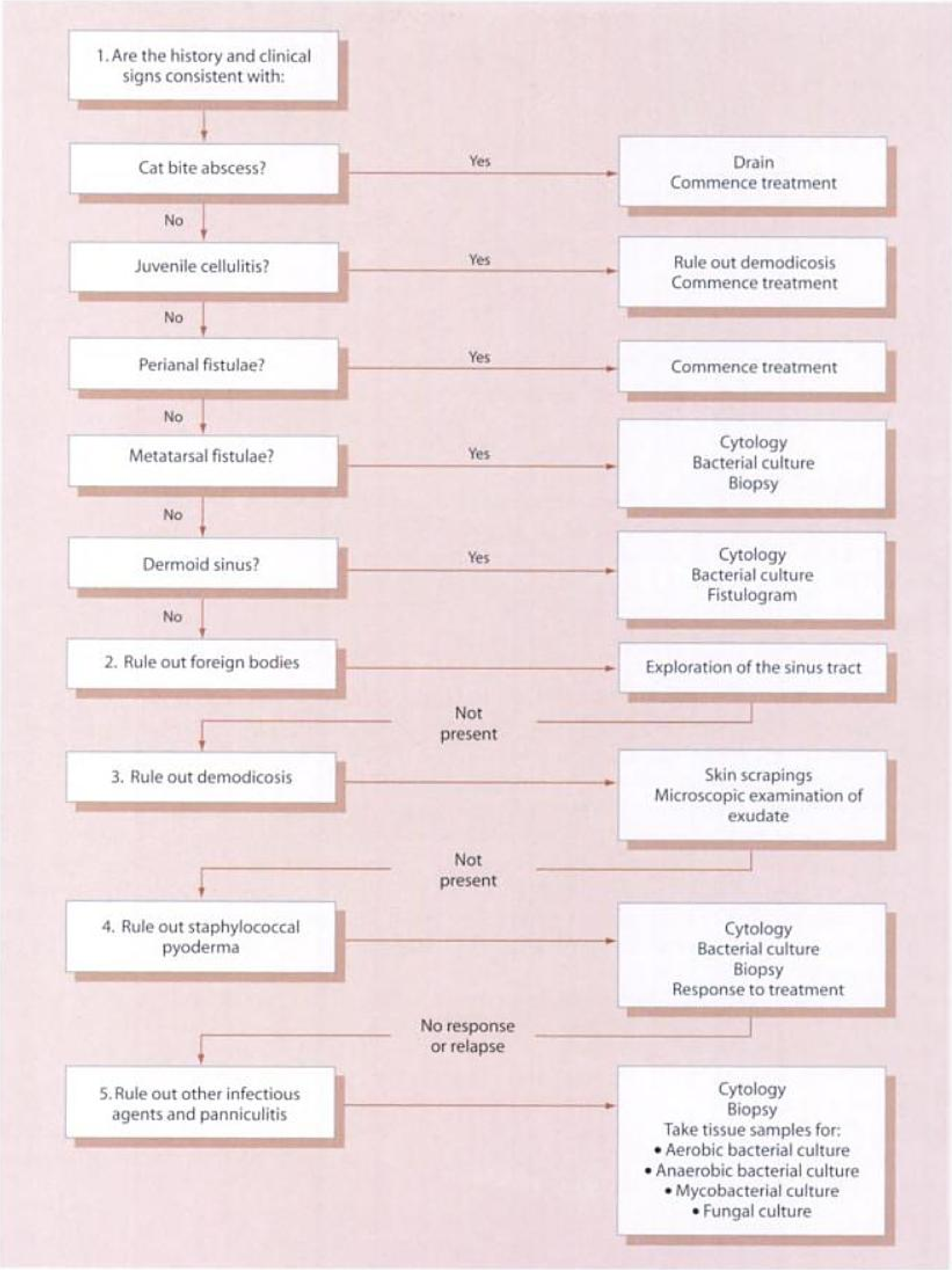


Figure 8.1 General diagnostic approach to draining tracts and non-healing wounds

9

Erosive and ulcerative diseases

Differential diagnosis

Erosions and ulcers are caused by diseases that damage the epidermis, dermo-epidermal junction or underlying dermis (trauma, autoimmune and immune-mediated diseases, deep infections, neoplasia, burns, congenital diseases). Some of these conditions are common whereas others are extremely rare.

When investigating the possible causes of erosions and ulcers, it is useful to divide the differential diagnoses into categories based on their presenting patterns. Table 9.1 divides these distribution patterns into three groups: diseases causing focal areas of ulceration; diseases causing ulceration of the nasal planum and face; and diseases causing ulceration of mucocutaneous junctions or generalised lesions.

Clinical features of erosive and ulcerative diseases

Many of the diseases listed below have been described earlier in this book because they present with other clinical signs in addition to ulceration. Acute moist dermatitis, acral lick dermatitis, and the eosinophilic granuloma complex are manifestations of pruritic skin diseases and are described in Chapter 4. Demodicosis usually presents as alopecia and is described in Chapter 5. Demodicosis can also cause scaling (Chapter 6), pustules (Chapter 7), draining tracts (Chapter 8) and occasionally ulceration when secondary infection is present. Dermatomyositis usually causes alopecia as described in Chapter 5 but leads to ulceration in severe cases. Pemphigus foliaceus and pemphigus erythematosus usually cause pustular eruptions that become crusted (Chapter 7), but ulceration may occur in severe cases. Although uveodermatological syndrome can cause ulceration in severe cases, it usually presents as a depigmenting disease and is described in Chapter 10. Epitheliotropic lymphoma may present as a pruritic skin disease as described in Chapter 4, but alopecia and scaling (Chapters 5 and 6), depigmentation (Chapter 10), cutaneous nodules (Chapter 11) or ulceration can all occur. Deep bacterial infections usually lead to draining tracts (Chapter 8) but ulceration may be a feature, especially in the German shepherd dog. Feline cowpox virus infection has been described in Chapter 7 as a papular disease but is listed here because the lesions can become ulcerated in some cases.

Intertrigo (skin fold dermatitis)

Aetiology: An ulcerative dermatosis caused by friction between skin surfaces that rub together.

Table 9.1 Differential diagnoses for erosive and ulcerative diseases

Lesion distribution	Specific disease
Focal lesions	**Acute moist dermatitis (D)**
	Acral lick dermatitis (lick granuloma) (D)
	Eosinophilic granuloma complex (C)
	Demodicosis (D and C)
	Intertrigo (D and C)
	Burns (D and C)
	Neoplasia (D and C)
Facial and nasal lesions	**Squamous cell carcinoma (D and C)**
	Demodicosis (D and C)
	Cutaneous lupus (D and C)
	Pemphigus foliaceus (D and C)
	Pemphigus erythematosus (D)
	Dermatomyositis (D)
	Epitheliotropic lymphoma (D and C)
	Uveodermatological syndrome (D)
	Herpes virus infection (C)
	Mucocutaneous pyoderma (D)
	Cowpox virus infection (C)
Mucocutaneous or generalised lesions	**Deep bacterial infection (D)**
	Generalised demodicosis (D)
	Systemic or cutaneous lupus (D and C)
	Pemphigus vulgaris (D and C)
	Sub-epidermal blistering diseases (D)
	Erythema multiforme major (D and C)
	Toxic epidermal necrolysis (D and C)
	Vasculitis (D and C)
	Drug eruption (D and C)
	Epidermolysis bullosa (D and C)
	Candidiasis (D)

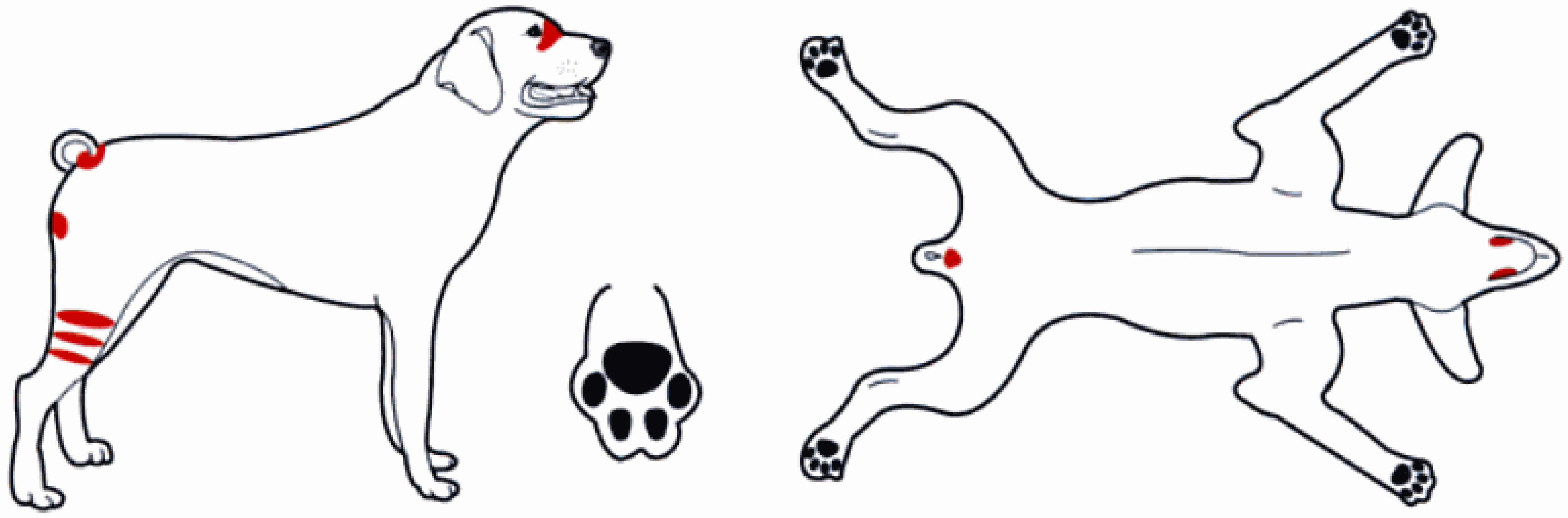
Those in bold would be most common in general practice. D = those diseases that may be seen in dogs, C = those diseases that may be seen in cats.

Predominant lesions:

Ulceration, exudation, surface infection, foul odour.

Lesion distribution:

Facial folds – Boston terrier, Bulldog, Pekinese, Pug, Persian cats
Lip folds – Cocker Spaniel, Springer Spaniel, St. Bernard
Vulval fold – Overweight bitches with a deeply recessed vulva
Body folds – Basset hound, Shar Pei
Tail fold – Boston terrier, Bulldog, Pug



Appropriate diagnostic tests:

Direct examination
Cytology

Burns

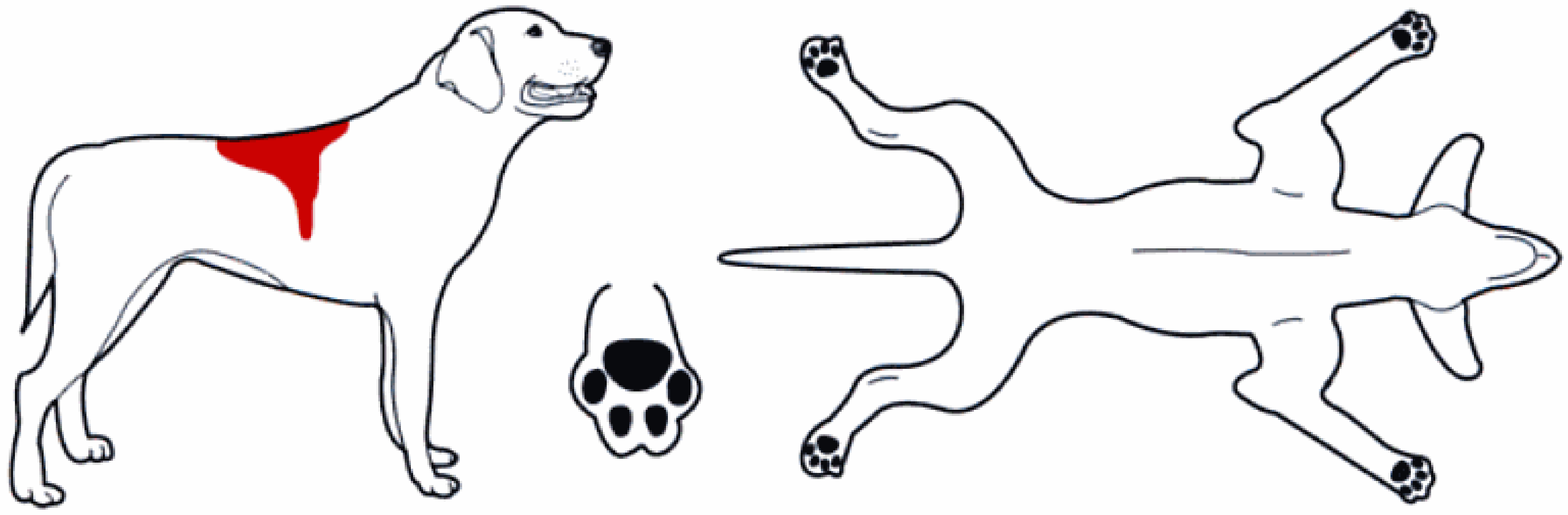
Aetiology: Necrosis of the skin caused by excessive heat. Can be caused by boiling water, car exhausts, surgical heat pads etc.

Predominant lesions:

Ulcers if severe.

Lesion distribution:

At the points of heat application.



Appropriate diagnostic tests:

Historical diagnosis
Biopsy

Neoplasia

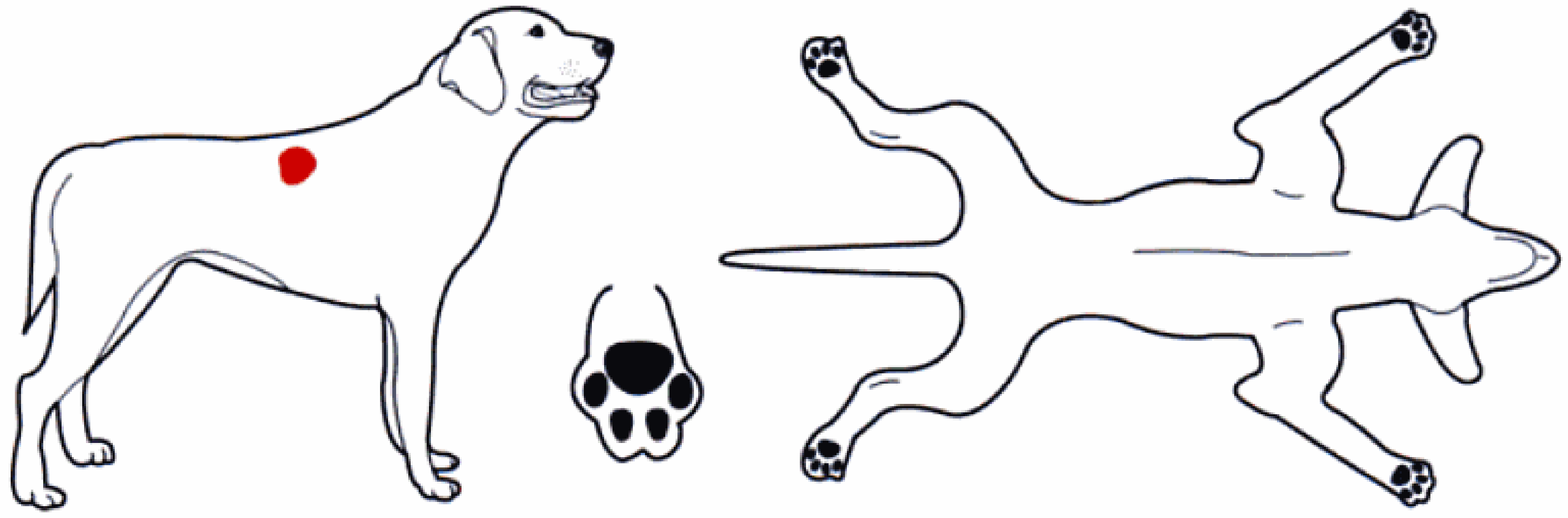
Aetiology: A variety of cutaneous neoplasms.

Predominant lesions:

Nodules, tumours, ulceration.

Lesion distribution:

Anywhere.



Appropriate diagnostic tests:

Cytology
Biopsy

Squamous cell carcinoma

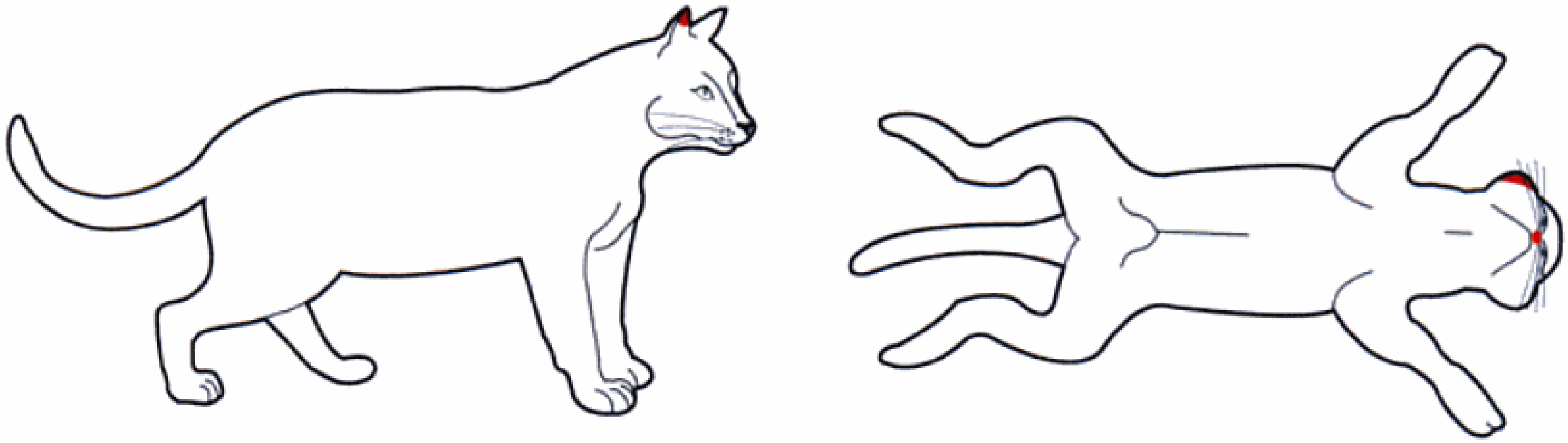
Aetiology: Neoplastic transformation of the epidermis often induced by sunlight.

Predominant lesions:

Destructive ulcerative lesions.

Lesion distribution:

Nasal planum, pinnae.



Appropriate diagnostic tests:

Cytology
Biopsy

Herpes virus infection

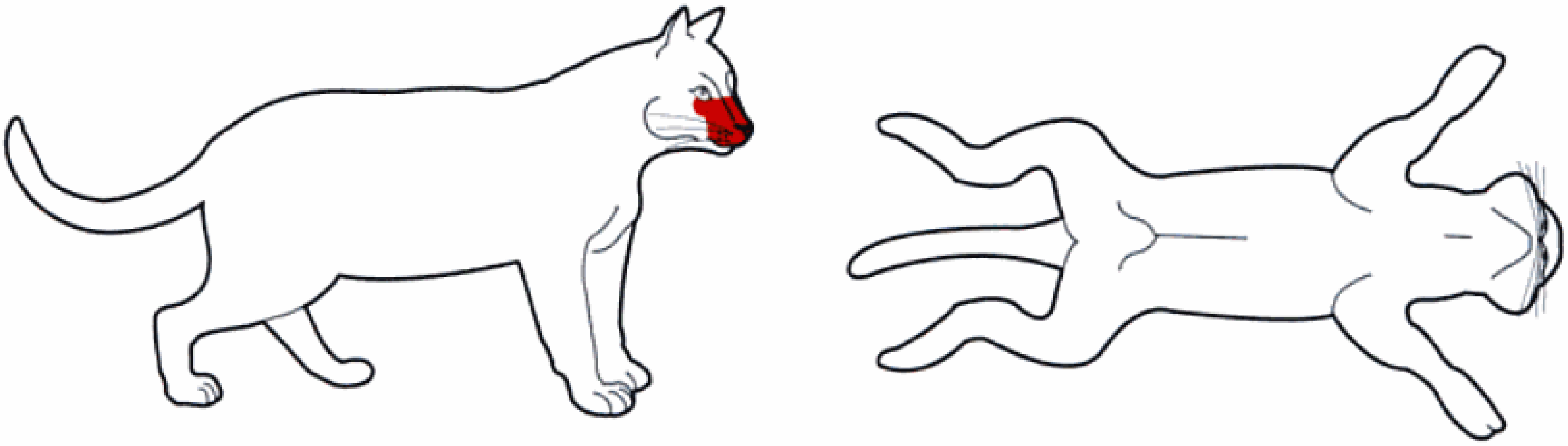
Aetiology: Infection of keratinocytes with feline herpes virus.

Predominant lesions:

Ulcerative lesions.

Lesion distribution:

Face.



Appropriate diagnostic tests:

Biopsy
Virus serology
Virus isolation

Mucocutaneous pyoderma

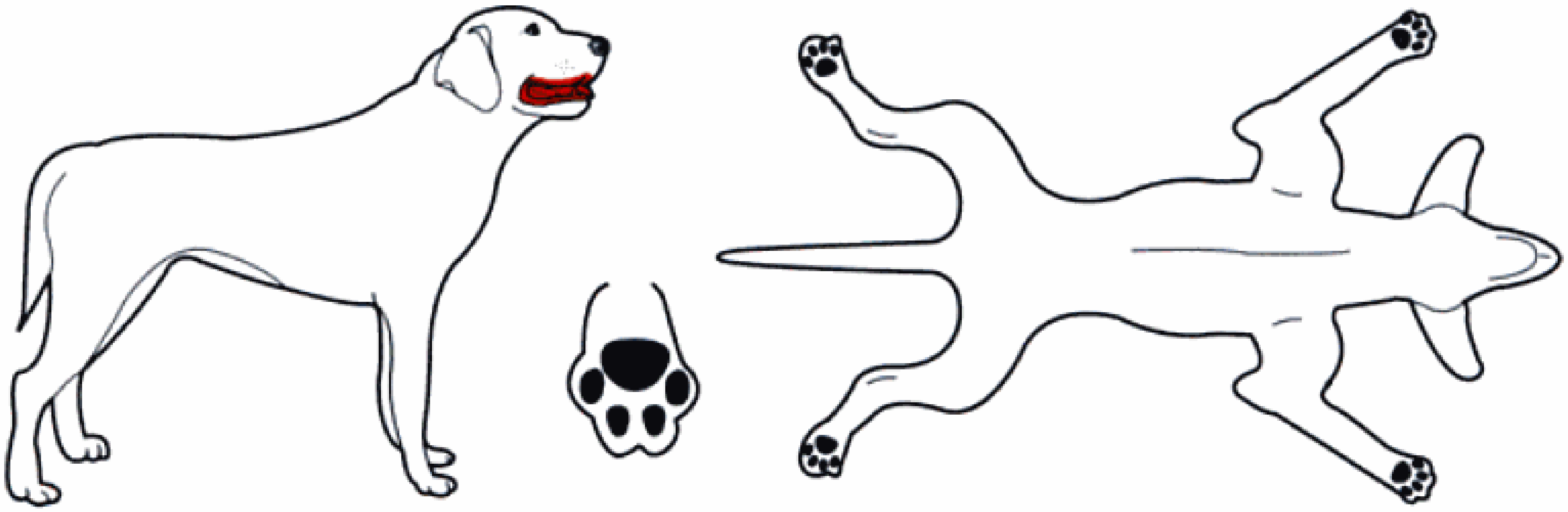
Aetiology: A staphylococcal infection of the lips that may mimic autoimmune diseases and epitheliotropic lymphoma. Most common in German shepherd dogs.

Predominant lesions:

Ulcers.

Lesion distribution:

Mucocutaneous junctions of the lips.



Appropriate diagnostic tests:

Cytology
Biopsy

Cutaneous and systemic lupus erythematosus

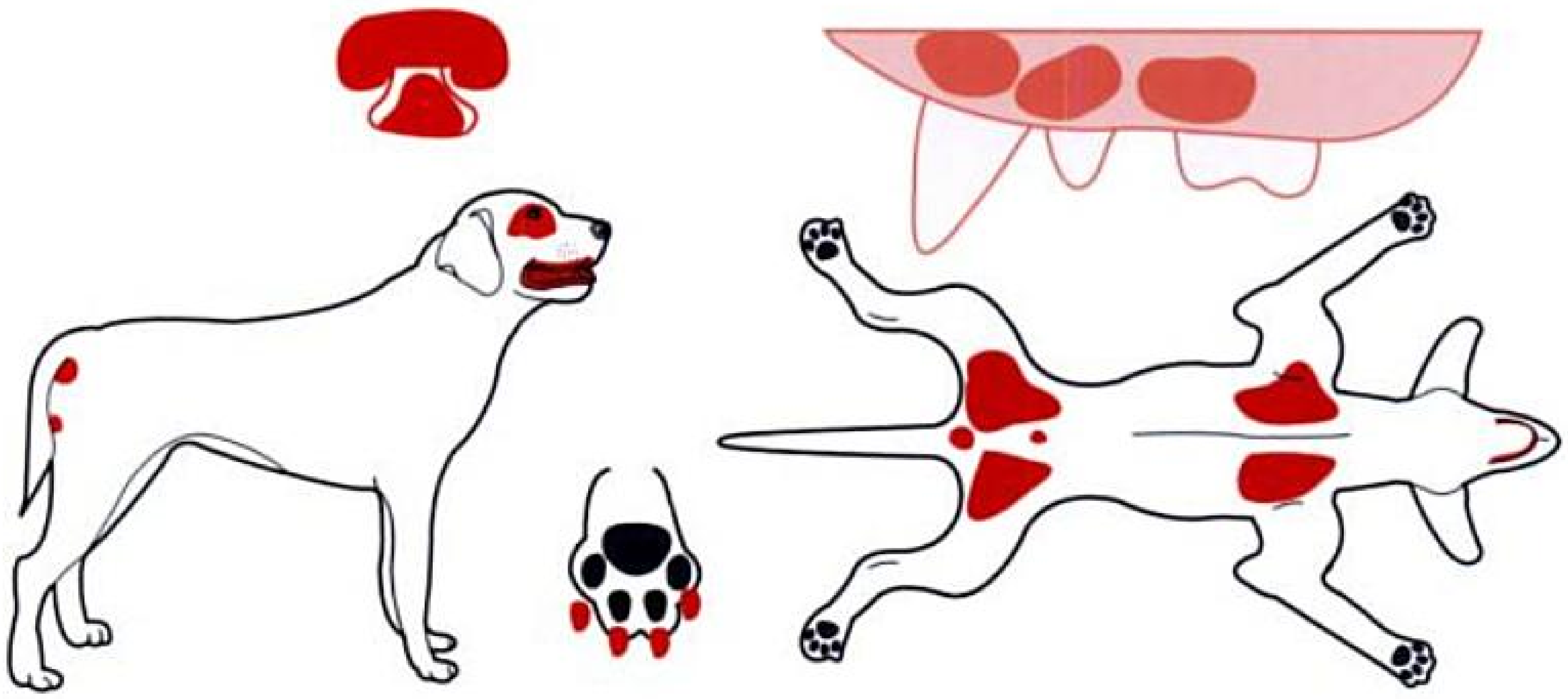
Aetiology: A rare disease caused by autoimmunity to various nuclear and cytoplasmic antigens, including DNA. The disease may affect the skin only (CLE), various internal organs (SLE), or both.

Predominant lesions:

Ulcers, crusts, alopecia, scaling, depigmentation. Rarely vesicles.

Lesion distribution (CLE):

- Photoaggravated facial lupus (previously DLE) – nasal planum, bridge of nose, face
- Oral cavity lupus – buccal mucosa, palate
- Mucocutaneous lupus – mucocutaneous junctions
- Ulcerative cutaneous lupus of Collies and Shetland Sheepdogs – ventral abdomen, axillae, mucocutaneous junctions
- Lupoid dermatosis of the German Shorthaired Pointer – generalised (see Chapter 6)
- Lupoid onychodystrophy – claws



Systemic signs (only occur in SLE):

- Immune-mediated haemolytic anaemia
- Immune-mediated thrombocytopaenia
- Sterile polyarthritis
- Glomerulonephritis

Appropriate diagnostic tests:

Haematology
Biochemistry
Urinalysis
Antinuclear antibody test
Biopsy

Pemphigus vulgaris

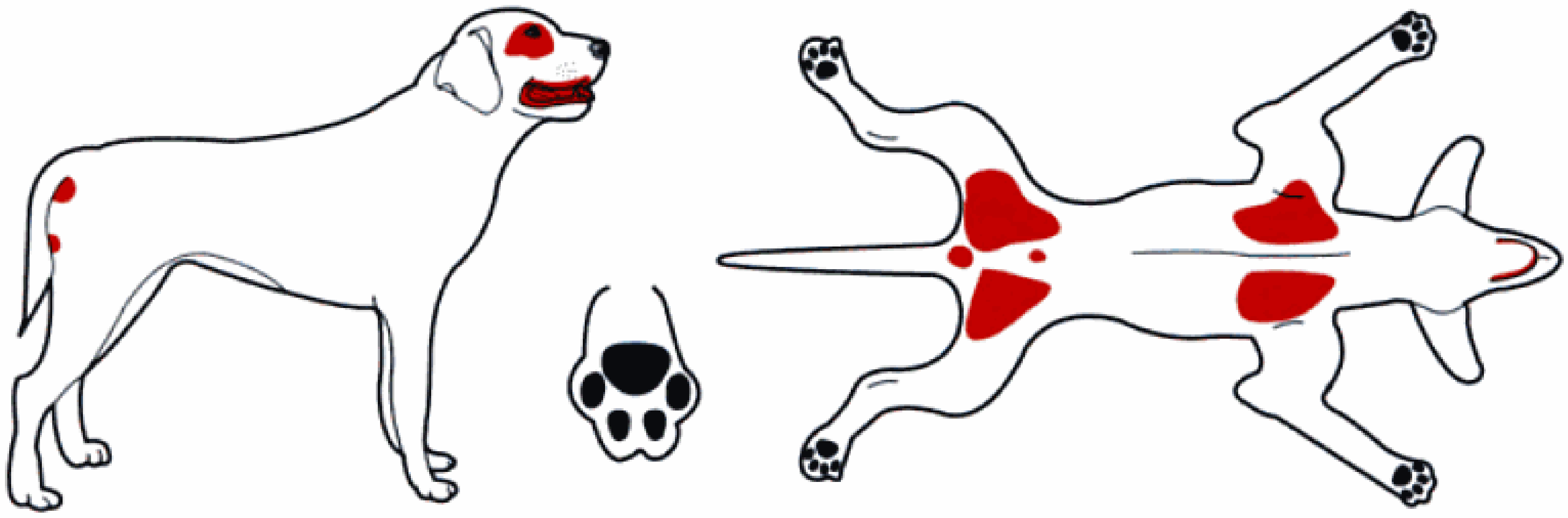
Aetiology: A very rare autoimmune skin disease in which autoantibodies attack intercellular adhesion molecules in the basal layers of the epidermis leading to acantholysis (separation of keratinocytes).

Predominant lesions:

Lesions begin as vesicles but nearly always present as ulcers.

Lesion distribution:

Oral cavity, mucocutaneous junctions, groin, axillae.



Appropriate diagnostic tests:

Biopsy

Sub-epidermal blistering diseases (bullous pemphigoid, mucous membrane pemphigoid, linear IgA bullous dermatosis, epidermolysis bullosa acquisita)

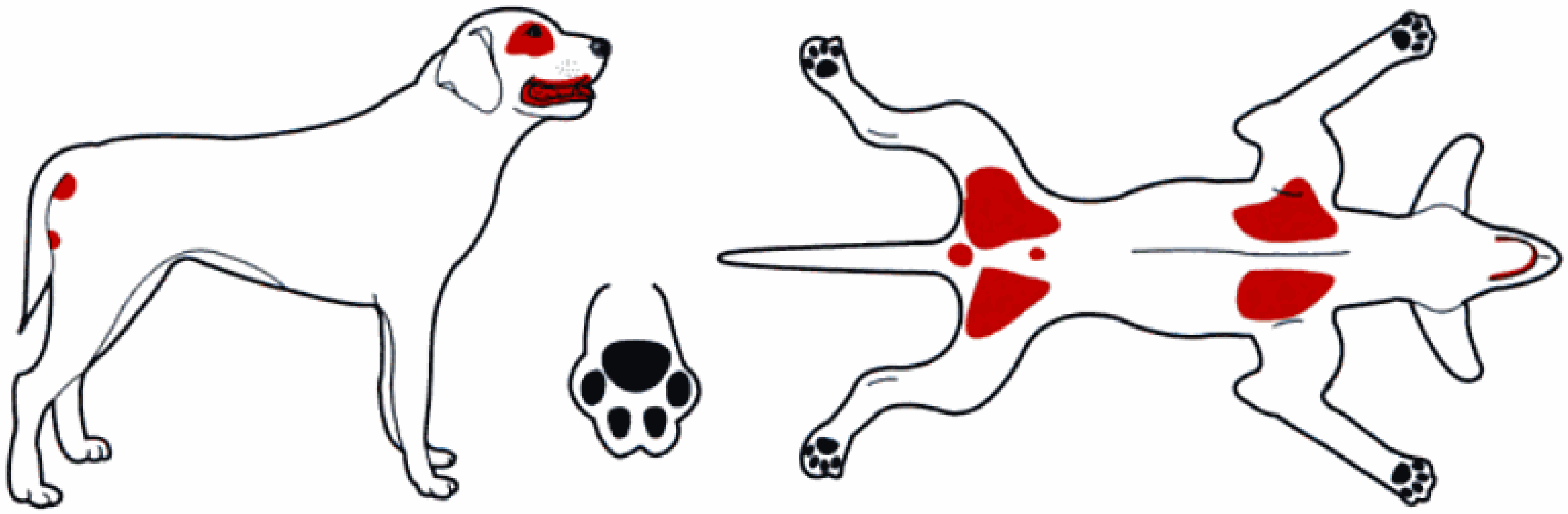
Aetiology: A rare group of autoimmune skin diseases in which autoantibodies attack various adhesion molecules in the basement membrane zone (dermo-epidermal junction).

Predominant lesions:

Lesions begin as vesicles but nearly always present as ulcers.

Lesion distribution:

Oral cavity, mucocutaneous junctions, groin, axillae.



Appropriate diagnostic tests:

Biopsy

Erythema multiforme major (Stevens Johnson's syndrome)

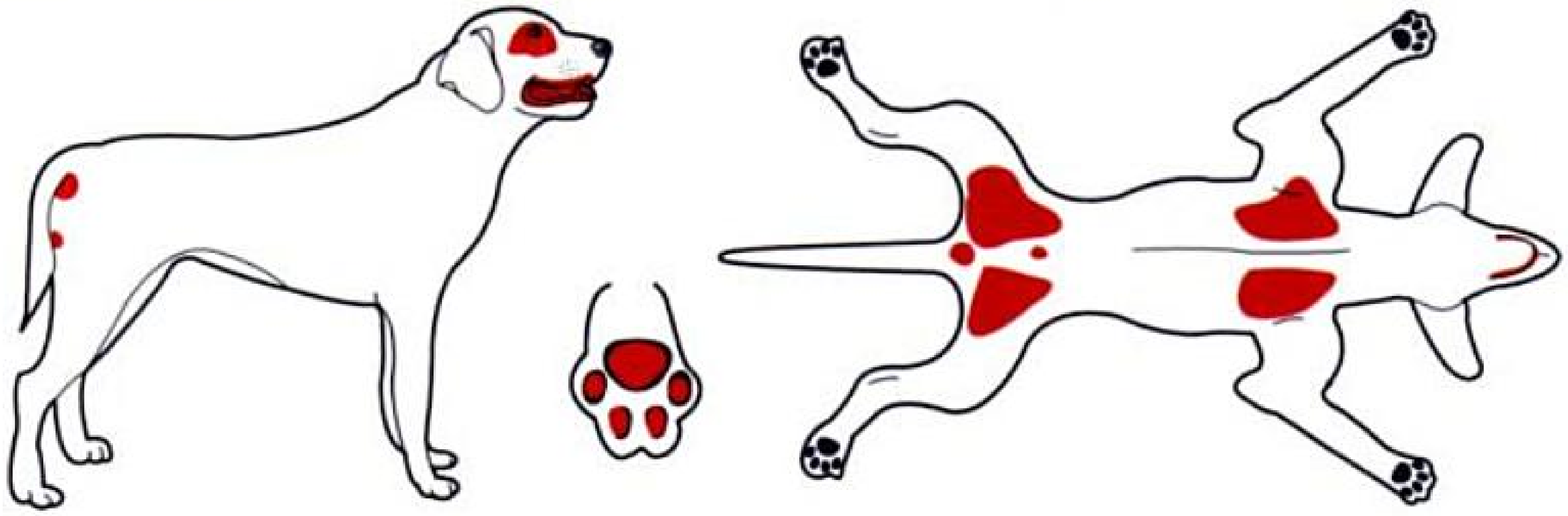
Aetiology: A sudden onset, immune-mediated skin disease that can be associated with adverse reactions to drugs or viral infections. Erythema multiforme can also lead to a less severe macular eruption (described in Chapter 7).

Predominant lesions:

Ulcers.

Lesion distribution:

Mucocutaneous junctions, groin, axillae, pressure points, footpads but can be more generalised.



Appropriate diagnostic tests:

Biopsy

Toxic epidermal necrolysis

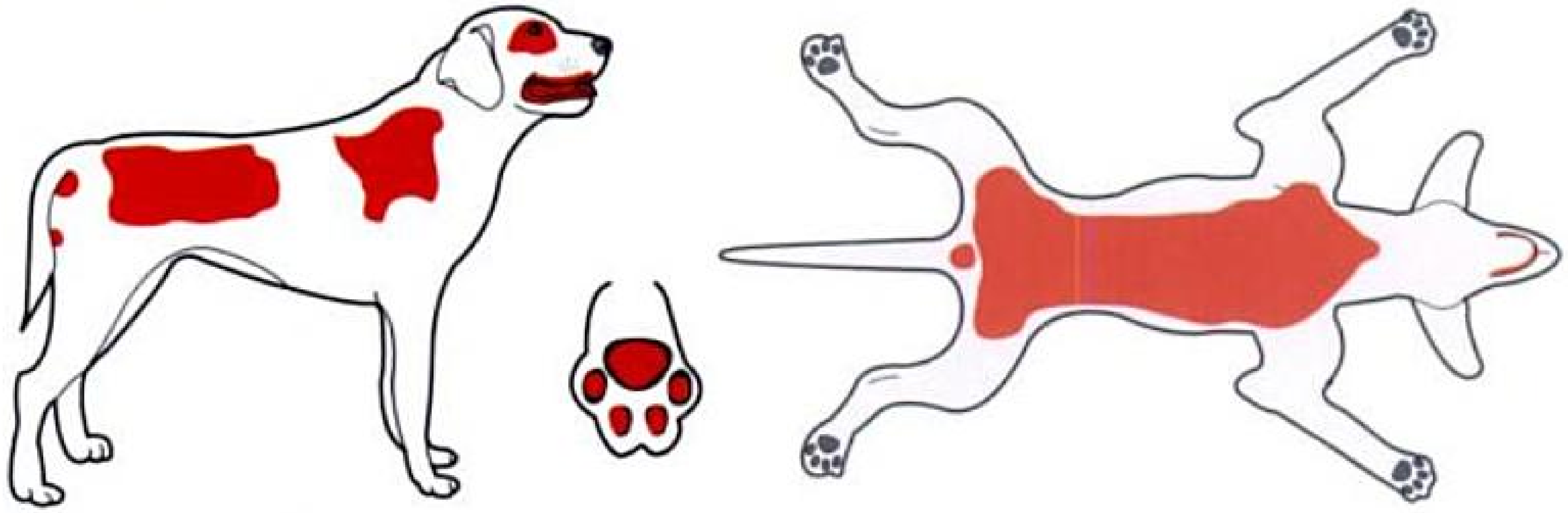
Aetiology: A sudden onset, widespread skin disease in which the epidermis sloughs off leaving skin that resembles a burn. May be associated with adverse reactions to drugs or severe systemic illnesses.

Predominant lesions:

Ulcers.

Lesion distribution:

Generalised.



Appropriate diagnostic tests:

Biopsy

Vasculitis

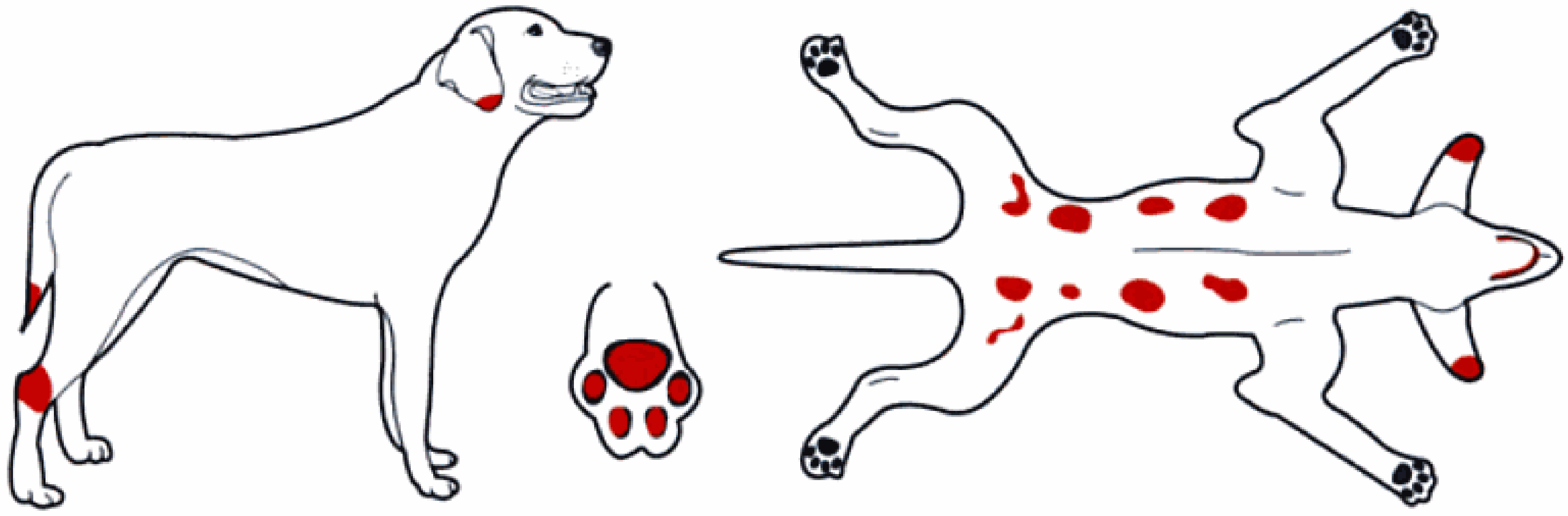
Aetiology: Immune-mediated inflammation of cutaneous blood vessels. Inflamed and thrombosed vessels can lead to cutaneous infarction and subsequent ulceration. May be associated with infections, systemic diseases, adverse reactions to drugs, lupus, or idiopathic. Vasculitis can also lead to purpura (described in Chapter 7).

Predominant lesions:

Punched out ulcers, oedema, purpura.

Lesion distribution:

Most obvious on the ventrum, extremities and over pressure points.



Appropriate diagnostic tests:

Diascopy (pressing a glass slide onto the skin to see if it blanches)
Biopsy

Drug eruption

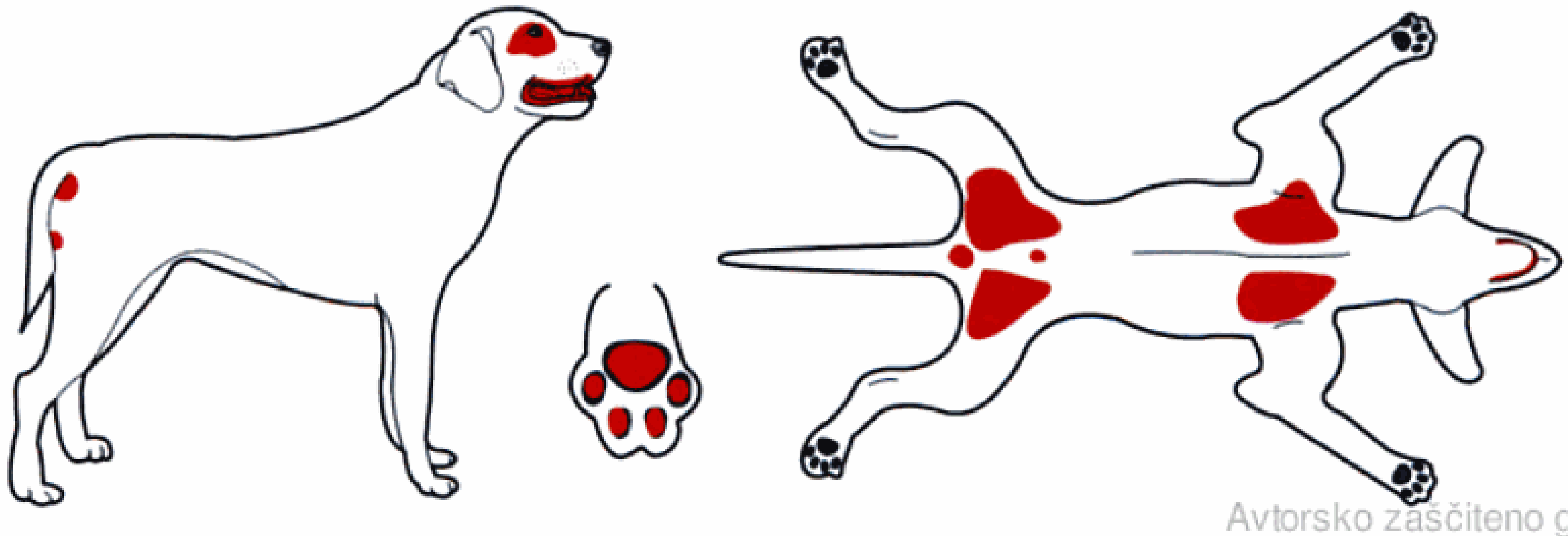
Aetiology: An adverse reaction to a drug leading to a cutaneous eruption. Drug eruptions occur sporadically, take many forms and will appear in other chapters of this book. Drug eruptions may cause erythema multiforme, toxic epidermal necrolysis, vasculitis, or other forms of ulcerative lesions.

Predominant lesions:

Ulcers.

Lesion distribution:

Anywhere, depending on the form encountered.



Appropriate diagnostic tests:

Biopsy

Epidermolysis bullosa

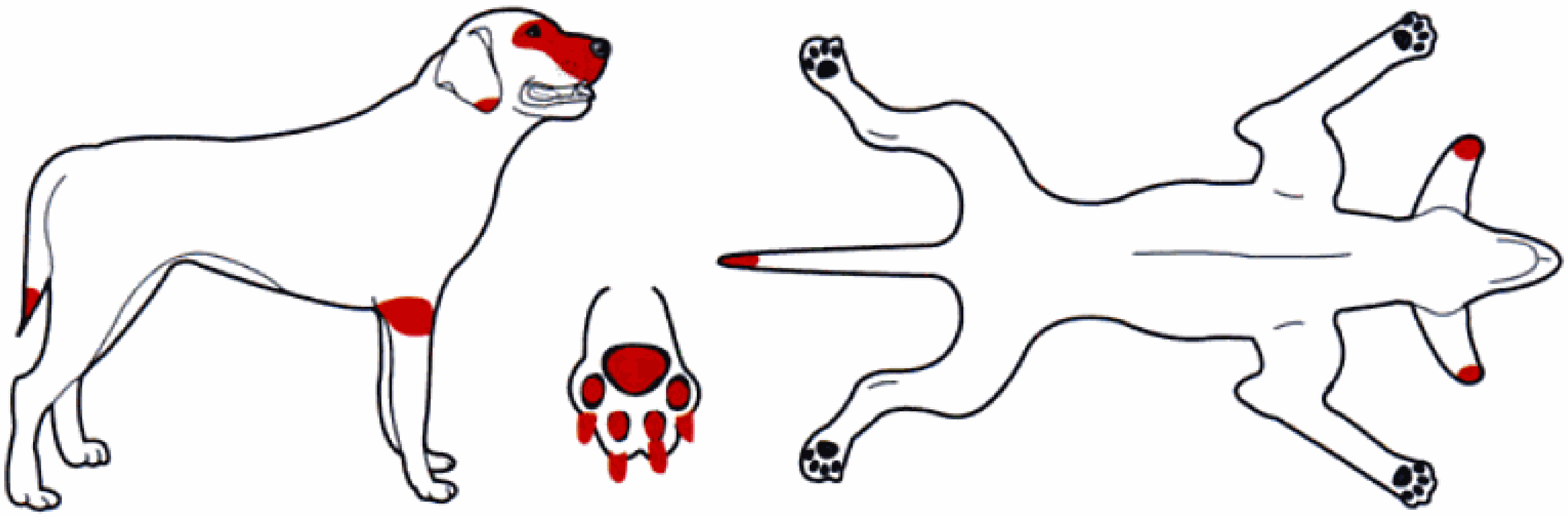
Aetiology: A group of congenital diseases caused by mutations in keratin or anchoring filament genes, resulting in separation of the epidermis from the dermis.

Predominant lesions:

Ulcers.

Lesion distribution:

Oral cavity, footpads, claws, pressure points, face.



Appropriate diagnostic tests:

Biopsy

Candidiasis

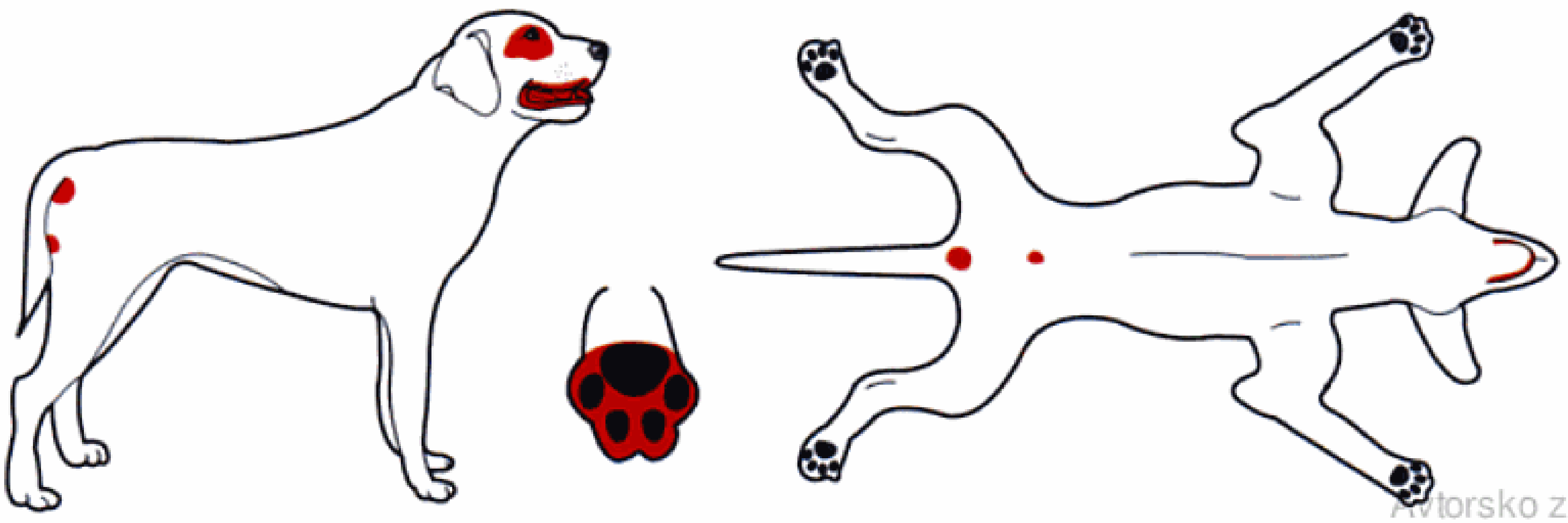
Aetiology: Cutaneous or mucous membrane infection with *Candida albicans*. Usually secondary to immunosuppressive diseases or drugs, or excessive antibiotic therapy.

Predominant lesions:

Ulcers covered in an adherent, whitish, gelatinous substance.

Lesion distribution:

Oral cavity, mucous membranes, mucocutaneous junctions, interdigital skin.

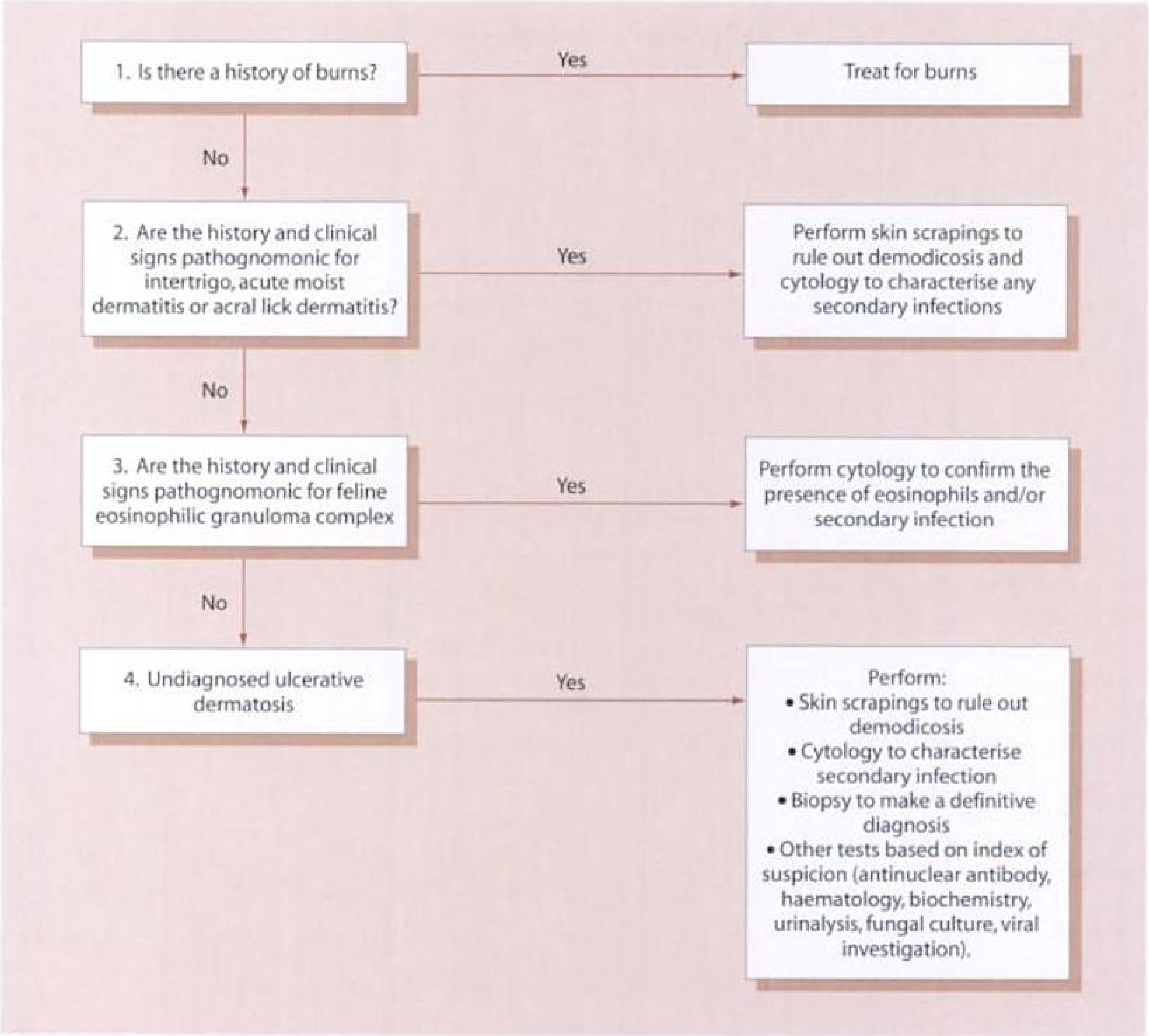


Appropriate diagnostic tests:

Cytology
Fungal culture
Biopsy

General diagnostic approach to erosive and ulcerative diseases

Ulcerative diseases can be severe and, in some cases, life-threatening. Hence, when presented with ulcerative lesions, it is critical that the clinician makes a swift and accurate diagnosis. There is no place for a protracted course of investigation that may be appropriate for some other presenting problems such as pruritus or scaling. Figure 9.1 shows a diagnostic algorithm that can be used to investigate cases of cutaneous ulceration.



1. Is there a history of burns?

Yes → Treat for burns

No ↓

2. Are the history and clinical signs pathognomonic for intertrigo, acute moist dermatitis or acral lick dermatitis?

Yes → Perform skin scrapings to rule out demodicosis and cytology to characterise any secondary infections

No ↓

3. Are the history and clinical signs pathognomonic for feline eosinophilic granuloma complex

Yes → Perform cytology to confirm the presence of eosinophils and/or secondary infection

No ↓

4. Undiagnosed ulcerative dermatosis

Yes → Perform:

- Skin scrapings to rule out demodicosis
- Cytology to characterise secondary infection
- Biopsy to make a definitive diagnosis
- Other tests based on index of suspicion (antinuclear antibody, haematology, biochemistry, urinalysis, fungal culture, viral investigation).

Figure 9.1 General diagnostic approach to ulcerative lesions

10

Pigmentary changes

Differential diagnosis

Pigmentation changes in the skin can be divided into those conditions that cause hyperpigmentation (Table 10.1) and those that cause hypopigmentation (Table 10.2).

Hyperpigmentation is seen most commonly following skin inflammation and with hormonal dermatoses. With these conditions, other presenting problems such as pruritus or alopecia are usually present concurrently.

Hypopigmentation can be caused by a congenital lack of melanin or diseases that lead to melanocyte damage. If the disease targets melanocytes specifically, hypopigmentation may be the only sign. If the melanocytes are damaged by a disease that affects the whole dermo-epidermal junction, other signs such as ulceration may also be present.

Table 10.1 Differential diagnoses for hyperpigmentation.

General Category	Specific disease
Post-inflammatory	**Flea bite hypersensitivity (D)**
	Demodicosis (D)
	Staphylococcal pyoderma (D)
	Malassezia dermatitis (D)
	Atopic dermatitis (D)
	Food intolerance (D)
Hormonal	**Hypothyroidism (D)**
	Hyperadrenocorticism (D)
	Testicular neoplasia (D)
	GH/adrenal sex hormone imbalance (D)
Neoplasia	**Melanoma, basal cell tumour (D and C)**
Congenital/Hereditary	**Acromelanism (C)**
	Lentigo (D and C)
	Acanthosis nigricans (D)
	Melanocytic naevi (D)

Those in bold would be most common in general practice. D = those diseases seen in dogs, C = those diseases seen in cats.

Table 10.2 Differential diagnoses for hypopigmentation

General category	Specific disease
Congenital/hereditary	Albinism (D and C)
	Chediak Higashi syndrome (C)
	Waardenburg-Klein syndrome (D and C)
Immune-mediated	Vitiligo (D and C)
	Uveodermatological syndrome (D)
	Lupus (D and C)
	Pemphigus complex (D and C)
Infectious	Leishmaniasis (D)
Nutritional	Zn deficiency (D)
	Rubber toxicity (D)
Neoplastic	Epitheliotropic lymphoma (D and C)
Environmental	Burns, cold, trauma, chemicals (D and C)
Idiopathic	**Nasal depigmentation (D)**
	Idiopathic leukotrichia (D)

Those in bold would be most common in general practice. D = those diseases seen in dogs, C = those diseases seen in cats.

Clinical features of skin diseases presenting with hyperpigmentation

Hyperpigmentation is usually caused by increased melanin pigment in the epidermis leading to a black or blackish-brown discoloration of the skin. This kind of hyperpigmentation is seen with most of the diseases listed in Table 10.1. Demodicosis leads to increased melanin in the dermis and causes a slate-grey hyperpigmentation.

The conditions causing post-inflammatory and hormonal hyperpigmentation have been described in Chapters 4 and 5 of this book because these diseases have more obvious presenting signs such as pruritus or alopecia. Testicular neoplasia may lead to diffuse hyperpigmentation of the ventrum and perineum in conjunction with alopecia (see Chapter 5) but a special form of macular melanosis seen with this disease is described here. This chapter also describes the conditions in which hyperpigmentation is the predominant sign.

Testicular neoplasia

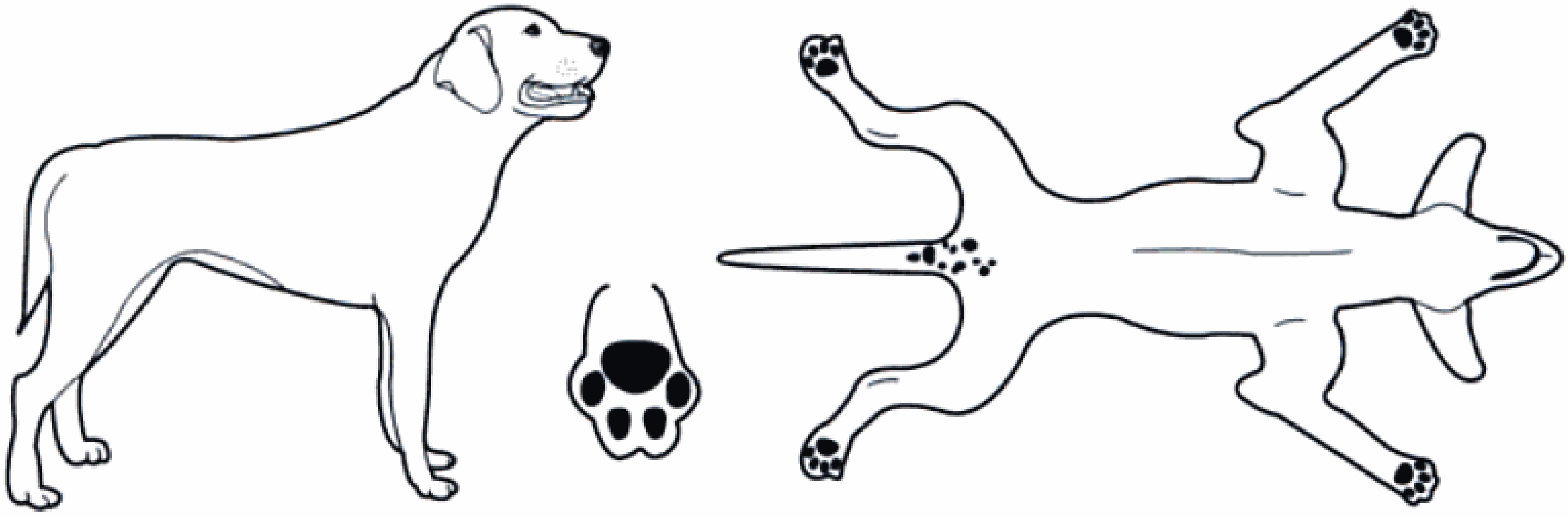
Aetiology: Melanisation affected by aberrant hormone production from testicular neoplasm.

Predominant lesions:

Hyperpigmented macules (macular melanosis).

Lesion distribution:

Anus, scrotum, prepuce, ventral proximal tail.



Appropriate diagnostic tests:

Abdominal radiography/ultrasound
Castration

Melanoma, basal cell tumours and melanocytic naevi

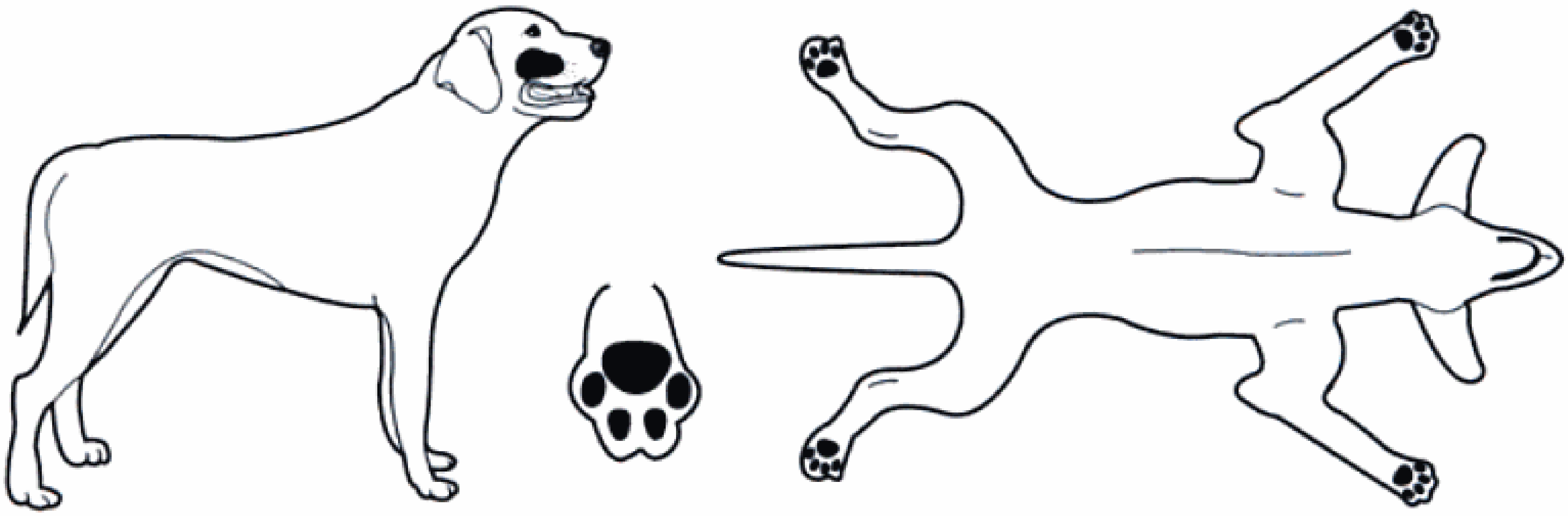
Aetiology: Neoplastic proliferation of melanocytes or basal cells, or congenital accumulation of melanocytes.

Predominant lesions:

Tumours.

Lesion distribution:

Melanoma – Head, neck, eyelids, muzzle, trunk, paws
Basal cell tumour – Eyelids, nasal planum, head, neck
Melanocytic naevi – Anywhere.



Appropriate diagnostic tests:

Cytology
Biopsy

Acromelanism

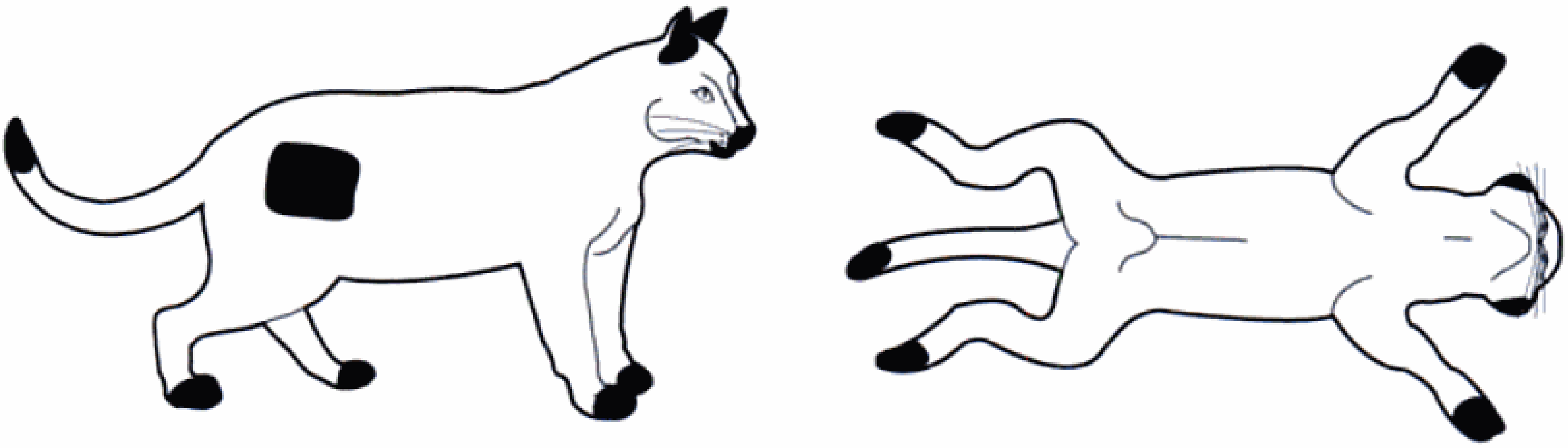
Aetiology: Dark hairs (points) are found on the extremities of some cats due to a temperature dependent effect on melanin synthesis. A similar darkening occurs after clipping due to a decrease in skin temperature.

Predominant lesions:

Dark hairs.

Lesion distribution:

At the site of hair clipping.



Appropriate diagnostic tests:

Clinical diagnosis

Lentigo

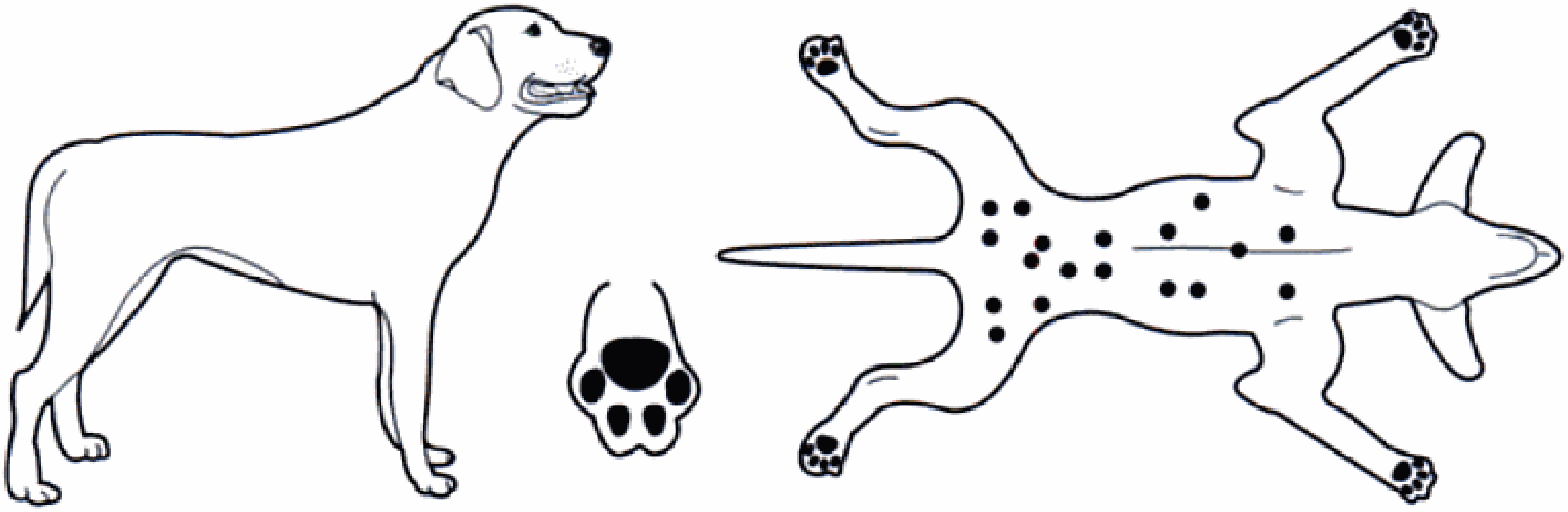
Aetiology: Genetically determined hypermelanosis seen in dogs and ginger cats.

Predominant lesions:

Sharply demarcated, intensely pigmented, hyperpigmented macules.

Lesion distribution:

Dog – ventral abdomen
Ginger cats – lips, nose, gingiva and eyelids



Appropriate diagnostic tests:

Biopsy

Acanthosis nigricans

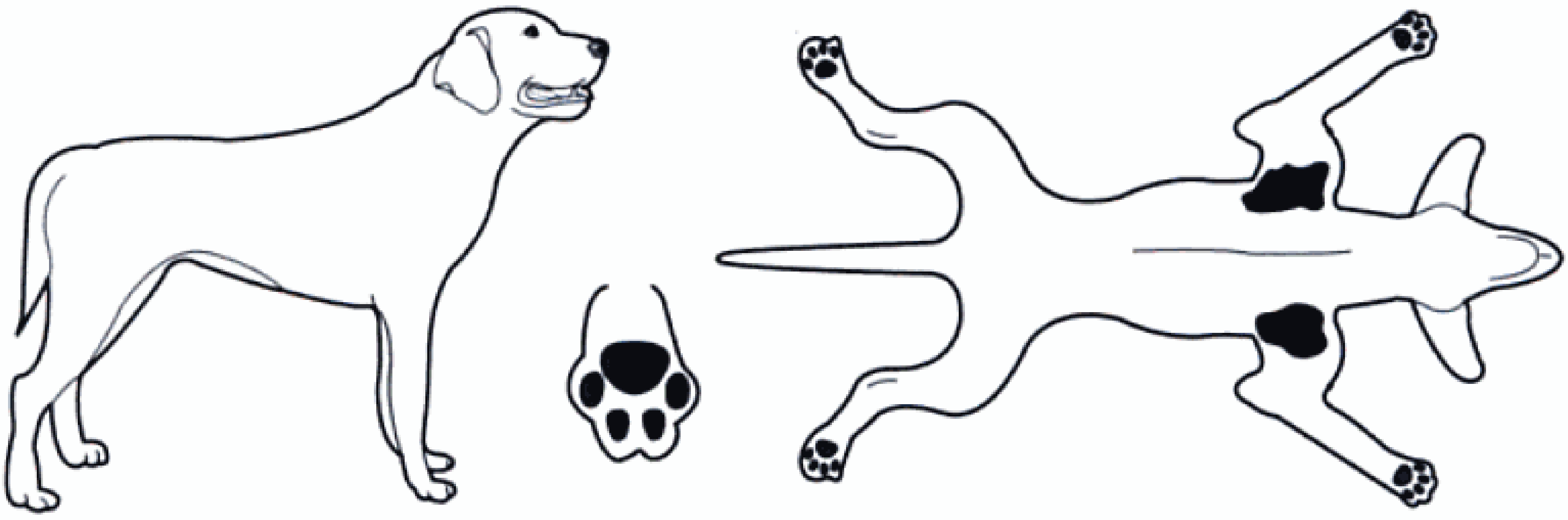
Aetiology: A condition described in older literature as a primary pigmentation disorder of Dachshunds. May actually be a reaction to chronic inflammation due to conditions such as allergies or Malassezia dermatitis.

Predominant lesions:

Hyperpigmentation.

Lesion distribution:

Axillae.



Appropriate diagnostic tests:

Tests to rule out inflammatory causes
Biopsy

Clinical features of skin diseases presenting with hypopigmentation

Hypopigmentation can affect the skin (leukoderma), the hair (leukotrichia) or both. This is commonly seen as a natural ageing process in dogs and is manifested by greying of the hair around the muzzle. This acquired leukotrichia is due to an age-related reduction in the number of cutaneous melanocytes. Hypopigmentation may also be seen in a number of disease states. For hypopigmentation to occur, there has to be an abnormality in melanin synthesis or abnormal/damaged melanocytes. In some of the diseases that cause hypopigmentation, the melanocytes are caught up in another disease process and hypopigmentation is not the only presenting sign. For example, in lupus (described in Chapter 8) and pemphigus (described in Chapter 7), the melanocytes are damaged by inflammation that targets the dermo-epidermal junction or epidermis. In leishmaniasis (Chapter 6), the melanocytes are damaged by infection of the epidermis with protozoal organisms whereas in epitheliotropic lymphoma (Chapter 4), the infiltrating lymphocytes damage keratinocytes and melanocytes concurrently. Zinc is required for normal melanin synthesis and zinc deficiency (described in Chapter 6) can lead to hypopigmentation in some cases. In all these cases, it is unlikely that the animal will be presented for hypopigmentation alone. The diagnosis should therefore be made by investigating the other presenting problems as described in earlier chapters. This chapter will describe the diseases in which hypopigmentation is the cardinal presenting sign.

Albinism

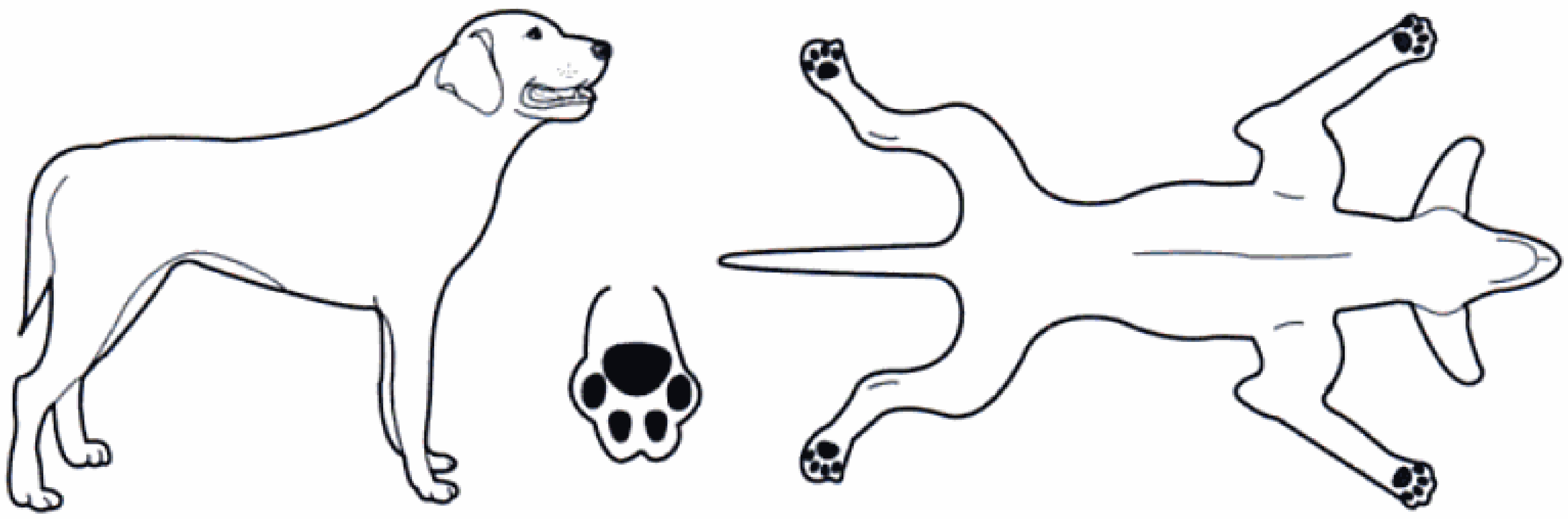
Aetiology: Extremely rare congenital disease in dogs and cats caused by a lack of melanin synthesis.

Predominant lesions:

Total lack of skin and hair pigment.

Lesion distribution:

Generalised.



Appropriate diagnostic tests:

Clinical diagnosis

Chediak Higashi syndrome

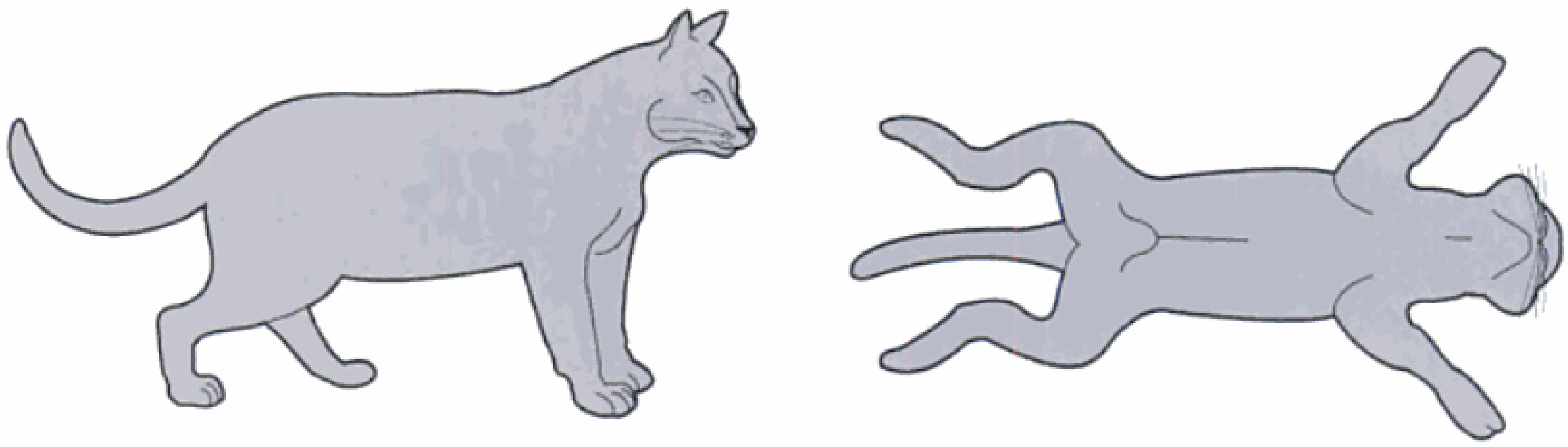
Aetiology: An inherited disorder of Persian cats with blue-smoke hair colour and yellow eyes.

Predominant lesions:

Dilute coat colour, non-pigmented retina, increased susceptibility to infections, bleeding disorders.

Lesion distribution:

Generalised.



Appropriate diagnostic tests:

Haematology (neutrophils and macrophages contain giant granules)
Trichogram
Biopsy

Waardenburg-Klein syndrome

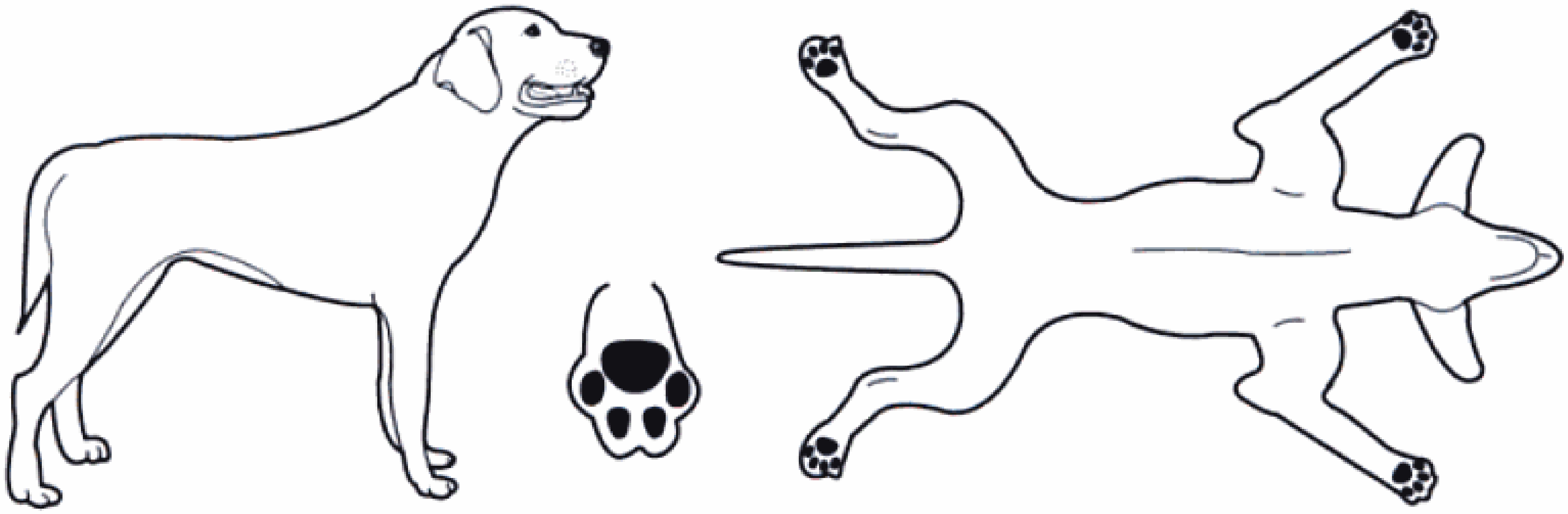
Aetiology: Rare congenital disease in dogs (Dalmatian, Collie, Bull terrier, Sealyham terrier) and cats caused by a defect in the migration and differentiation of melanocytes.

Predominant lesions:

Total lack of skin and hair pigment; blue eyes; and total deafness.

Lesion distribution:

Generalised.



Appropriate diagnostic tests:

Clinical diagnosis
Hearing tests

Vitiligo

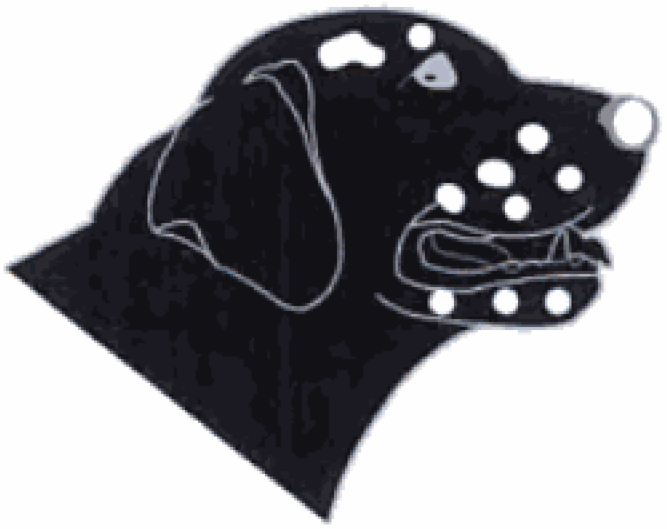
Aetiology: Immune-mediated destruction of melanocytes. Most common in Belgian Tervurens, Rottweilers and Doberman pinschers.

Predominant lesions:

Patches of symmetrical leukoderma and leukotrichia.

Lesion distribution:

Nose, lips, face, buccal mucosa, footpads, trunk.



Appropriate diagnostic tests:

Biopsy

Uveodermatological syndrome (Vogt-Koyanagi-Harada-like syndrome)

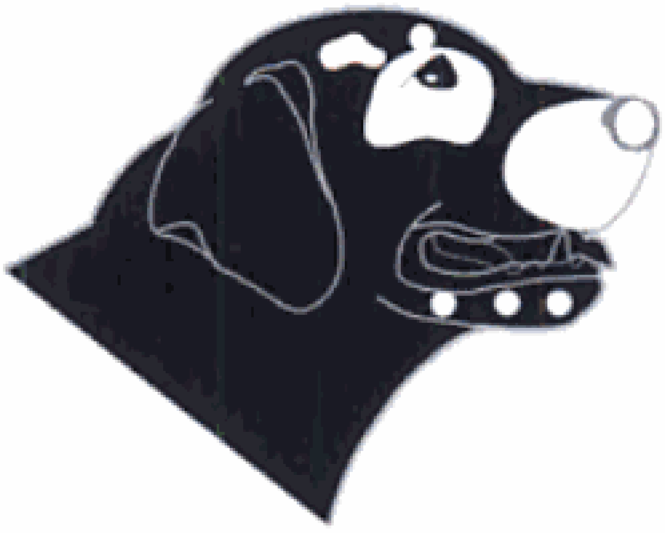
Aetiology: An autoimmune disease in which cutaneous and ocular melanocytes are damaged by autoantibodies and macrophages. Akitas, Chows, Samoyeds and Siberian huskies are predisposed.

Predominant lesions:

Leukotrichia, leukoderma, uveitis.

Lesion distribution:

Usually nose, lips, and eyelids. Occasionally affects other areas.



Appropriate diagnostic tests:

Biopsy

Rubber toxicity

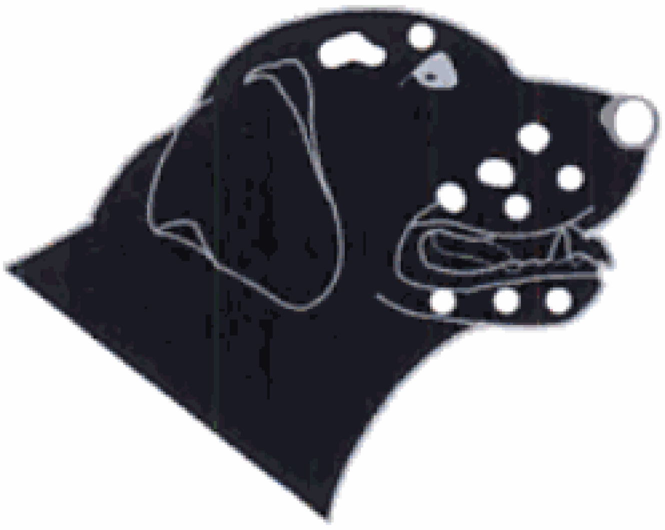
Aetiology: Melanocyte damage caused by ingestion of a toxic principle in rubber.

Predominant lesions:

Patches of symmetrical leukoderma and leukotrichia.

Lesion distribution:

Face, limbs, trunk.



Appropriate diagnostic tests:

Biopsy

Environmental damage (burns, cold, trauma, chemicals)

Aetiology: Damage to melanocytes caused by physical or chemical injury.

Predominant lesions:

Patches of leukoderma and leukotrichia.

Lesion distribution:

At the site of damage.

Appropriate diagnostic tests:

Historical diagnosis

Idiopathic nasal depigmentation (Dudley nose, snow nose)

Aetiology: Unknown aetiology but may be an ageing change, a form of vitiligo or related to photoperiod.

Predominant lesions:

Hypopigmentation.

Lesion distribution:

Nasal planum.

Appropriate diagnostic tests

None required

Idiopathic leukotrichia

Aetiology: Unknown but may be a form of vitiligo.

Predominant lesions:

Patches of leukotrichia.

Lesion distribution:

Head, trunk and legs.

Appropriate diagnostic tests:

Biopsy

General diagnostic approach to pigmentary changes

When animals are presented with hyperpigmentation or hypopigmentation it is important to determine if other clinical signs are also present. If the flowchart described in Chapter 3 is followed, most cases of pigmentation change will be investigated for other problems first. However, in certain cases, the clinician may be faced with the investigation of pigmentary abnormalities as the primary presenting problem. The flowcharts shown in Figures 10.1 and 10.2 describe the basic diagnostic approach to changes in pigmentation.

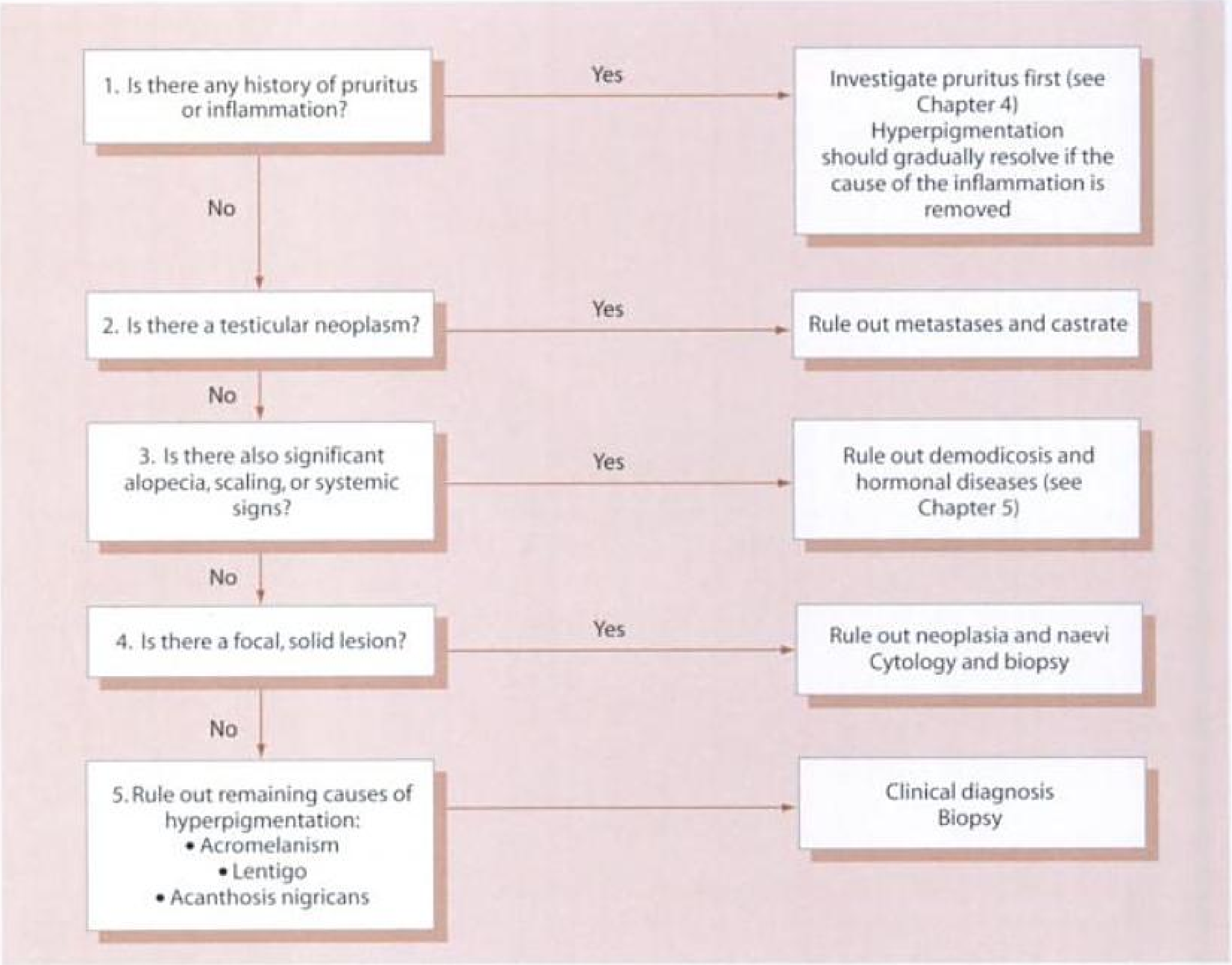

1. Is there any history of pruritus or inflammation?
Yes
Investigate pruritus first (see Chapter 4) Hyperpigmentation should gradually resolve if the cause of the inflammation is removed
No
2. Is there a testicular neoplasm?
Yes
Rule out metastases and castrate
No
3. Is there also significant alopecia, scaling, or systemic signs?
Yes
Rule out demodicosis and hormonal diseases (see Chapter 5)
No
4. Is there a focal, solid lesion?
Yes
Rule out neoplasia and naevi Cytology and biopsy
No
5. Rule out remaining causes of hyperpigmentation:
• Acromelanism
• Lentigo
• Acanthosis nigricans
Clinical diagnosis Biopsy

Figure 10.1 General diagnostic approach to hyperpigmentation

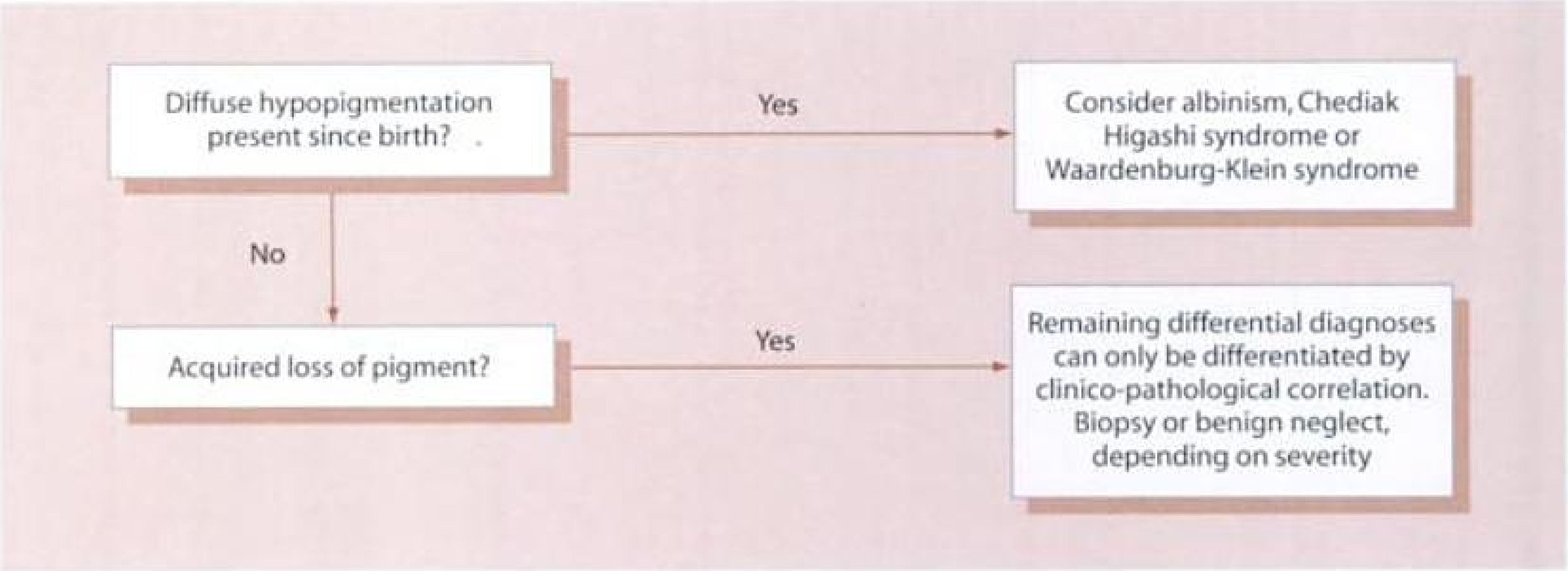

Diffuse hypopigmentation present since birth?
Yes
Consider albinism, Chediak Higashi syndrome or Waardenburg-Klein syndrome
No
Acquired loss of pigment?
Yes
Remaining differential diagnoses can only be differentiated by clinico-pathological correlation. Biopsy or benign neglect, depending on severity

Figure 10.2 General diagnostic approach to hypopigmentation

11

Lumps and swellings

Differential diagnosis

Cutaneous lumps and swellings are a common presenting sign in general practice. They are a source of great anxiety to owners because they are either visible or palpable, and may be due to neoplasia. Hence, when presented with such a problem, the clinician's role is to rapidly determine an accurate diagnosis and prognosis. Table 11.1 shows the possible causes of cutaneous swellings in dogs and cats based on the type of material that is found within them. This approach is useful because determining the contents of a lump is pivotal in making a specific diagnosis.

Clinical features of cutaneous lumps and swellings

It is not the intention of this chapter to provide detailed information about every skin tumour that has ever been recorded in dogs and cats. The aim is to provide an overview of the key features that allow the differential diagnoses to be prioritised. However, despite this information, the only way to reliably differentiate between the vast majority of the lumps described in Table 11.1 is to perform fine needle aspiration cytology and/or histopathology.

Table 11.1 Differential diagnoses for cutaneous lumps and swellings

Content of lump	Specific diagnosis
Blood	**Haematoma (D and C)**
	Vascular tumour (D and C)
Oedema	**Urticaria (D)**
	Angioedema (D)
Serum	**Seroma (D)**
Pus	**Abscess (D and C)**
	Deep bacterial infection (D and C)
	Mycetoma (D and C)
	Kerion (D)
	Deep fungal infection (D and C)
Inflammatory cells	Sterile nodular panniculitis (D and C)
	Sterile nodular granuloma and pyogranuloma (D and C)
	Cutaneous histiocytosis (D)
	Plasma cell pododermatitis (C)
Anuclear cellular and proteinaceous debris	**Cutaneous cyst** (D and C)
Fat	**Lipoma (D** and C)
Neoplastic or hyperplastic sebocytes	**Sebaceous gland hyperplasia/adenoma (D** and C)
	Tail gland hyperplasia (D)
Mast cells	**Mast cell tumour (D and C)**
Neoplastic epithelial cells	Papilloma (D and C)
	Squamous cell carcinoma (D and C)
	Basal cell tumour (D and **C)**
	Perianal gland tumour (D)
	Hair follicle tumours (D and C)
	Sweat gland tumours (D and C)
Neoplastic spindle cells	Fibroblast tumours (D and C)
	Haemangiopericytoma (D)
	Neural tumours (D and C)
	Vascular tumours (D and C)
Neoplastic round cells	Lymphoid tumours (D and C)
	Histiocytoma (D and C)
	Plasma cell tumour (D and C)
Melanocytes	Melanoma (D and C)
Calcium	Calcinosis cutis (D)
	Calcinosis circumscripta (D)
Mucin	Cutaneous mucinosis (D)
Hyperplastic tissue	Naevus (D and C)
	Dermatofibrosis (D)

Those in bold would be most common in general practice. D = those diseases seen in dogs, C = those diseases seen in cats.

Haematoma

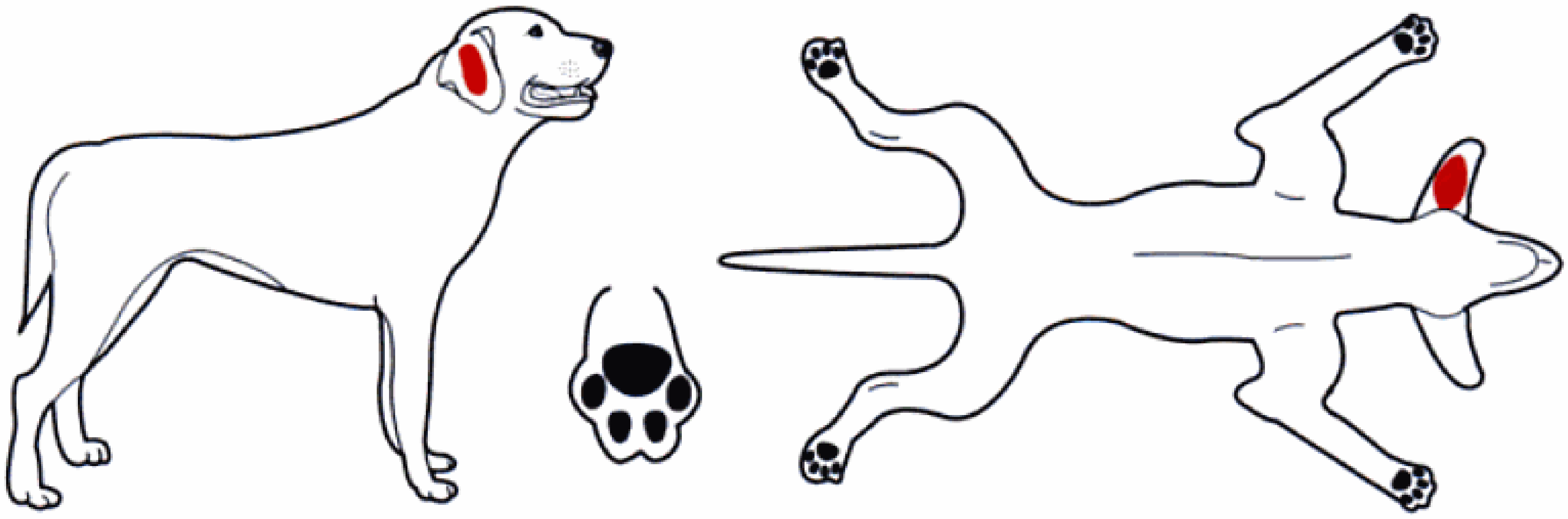
Aetiology: Trauma.

Predominant lesions:

Blood filled swelling.

Lesion distribution:

Most common on the pinna but can be anywhere.



Appropriate diagnostic tests:

Clinical diagnosis
Aspiration

Vascular tumours

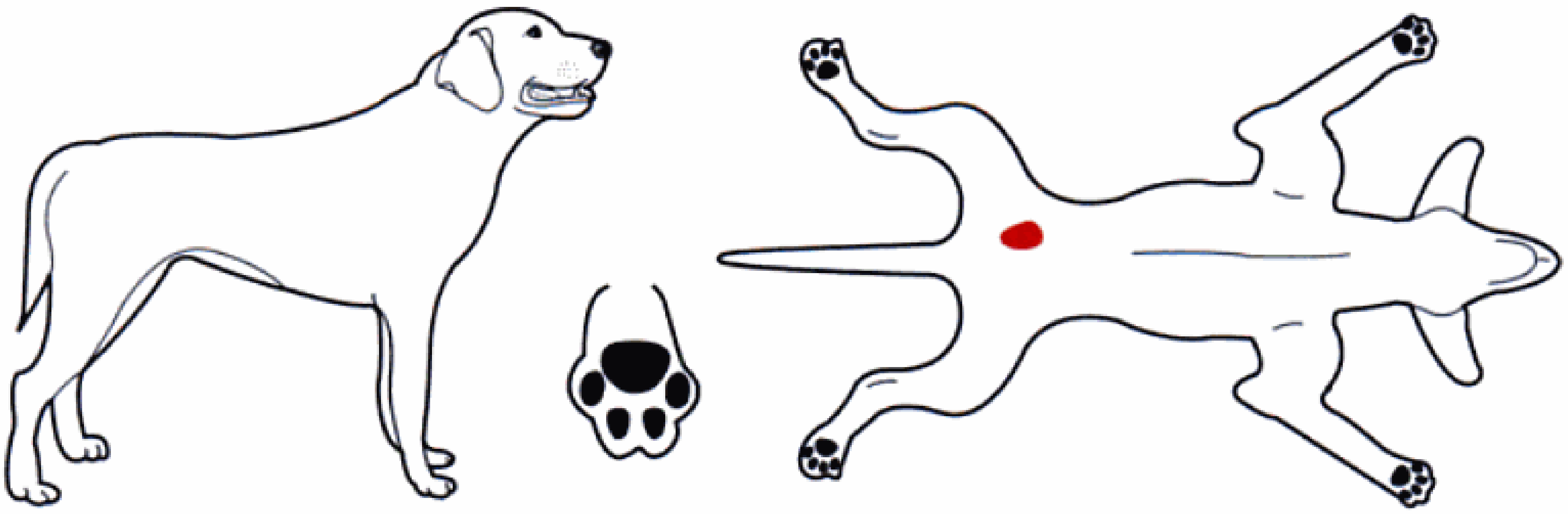
Aetiology: Neoplasm of blood vessel endothelial cells.

Predominant lesions:

Tumours.

Lesion distribution:

Anywhere.



Appropriate diagnostic tests:

Cytology
Biopsy

Urticaria

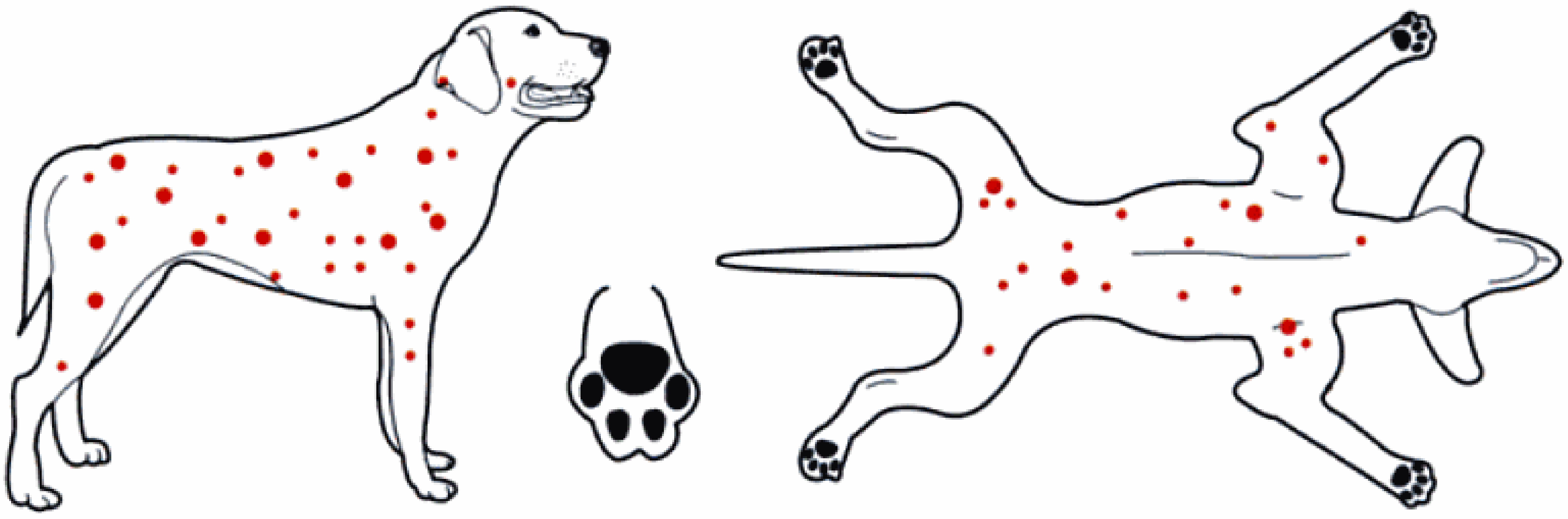
Aetiology: Acute allergic reaction (Type 1 hypersensitivity) to an ingested, inhaled, injected or contacted substance (food, drugs, insect bites, stings, plants, aeroallergens).

Predominant lesions:

Multiple wheals.

Lesion distribution:

Generalised.



Appropriate diagnostic tests:

Historical diagnosis
Restricted diet trial
Intradermal testing

Angioedema

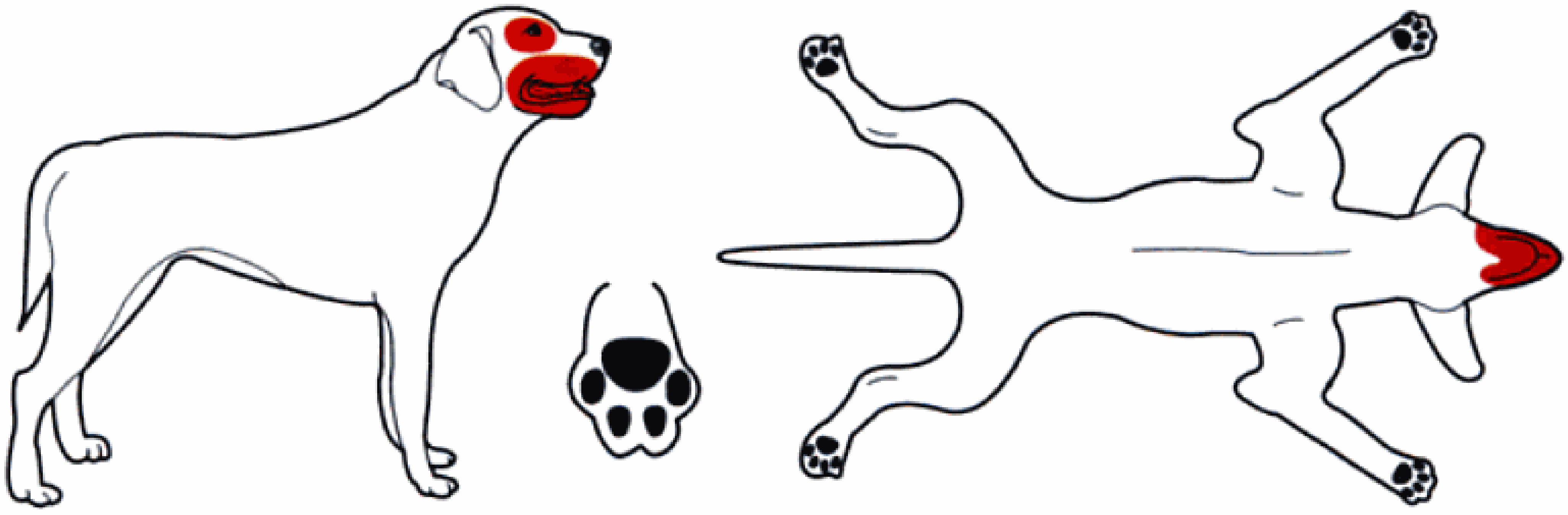
Aetiology: Acute allergic reaction (Type 1 hypersensitivity) to an ingested, inhaled, injected or contacted substance (food, drugs, insect bites, stings, plants, aeroallergens).

Predominant lesions:

Diffuse swelling.

Lesion distribution:

Periocular, lips, face



Appropriate diagnostic tests:

Historical diagnosis
Restricted diet trial
Intradermal testing

Seroma

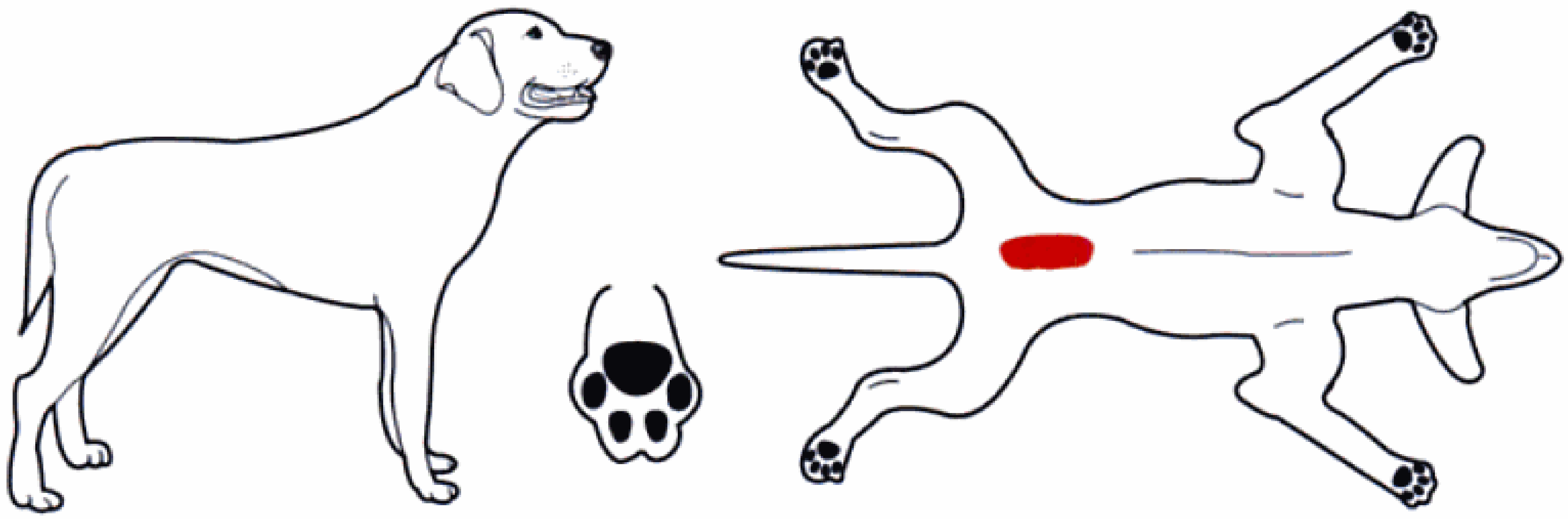
Aetiology: Tissue reaction to foreign material such as sutures.

Predominant lesions:

Swelling.

Lesion distribution:

At site of implants.



Appropriate diagnostic tests:

Historical diagnosis

Swellings containing pus (abscesses, deep bacterial infections, mycetomas, kerions, deep fungal infections)

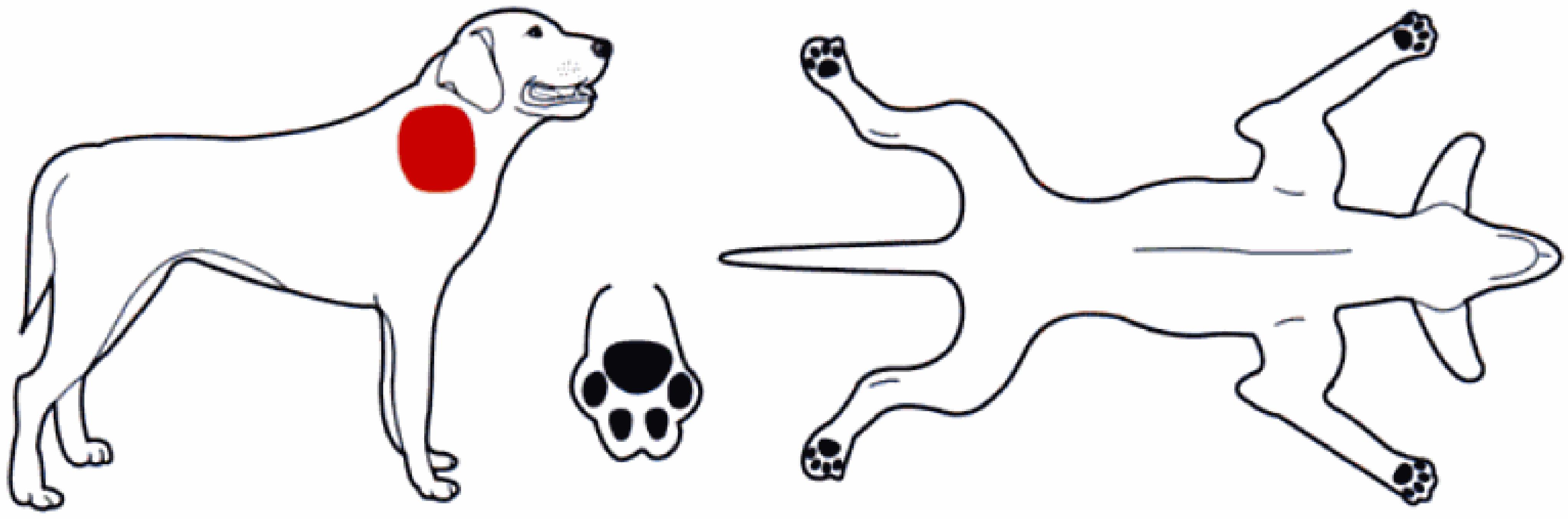
Aetiology: Subcutaneous infection.

Predominant lesions:

Swellings, draining tracts (see Chapter 9 for further details).

Lesion distribution:

Anywhere.



Appropriate diagnostic tests:

Cytology
Culture

Sterile nodular panniculitis

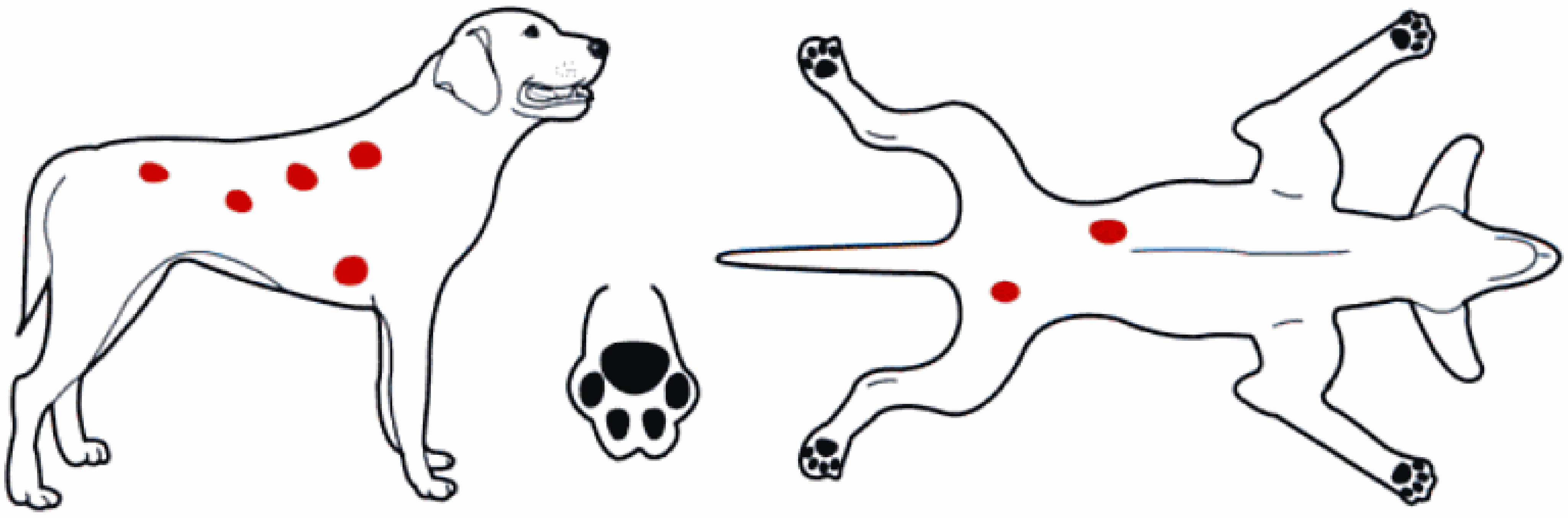
Aetiology: Inflammation of the subcutaneous fat of unknown aetiology (see also Chapter 9 for other causes of panniculitis).

Predominant lesions:

Nodules, draining tracts (exudate is usually oily).

Lesion distribution:

Usually over the trunk.



Appropriate diagnostic tests:

Cytology
Biopsy
Tests to rule out infectious and underlying causes

Sterile nodular granuloma and pyogranuloma (may be a form of histiocytosis)

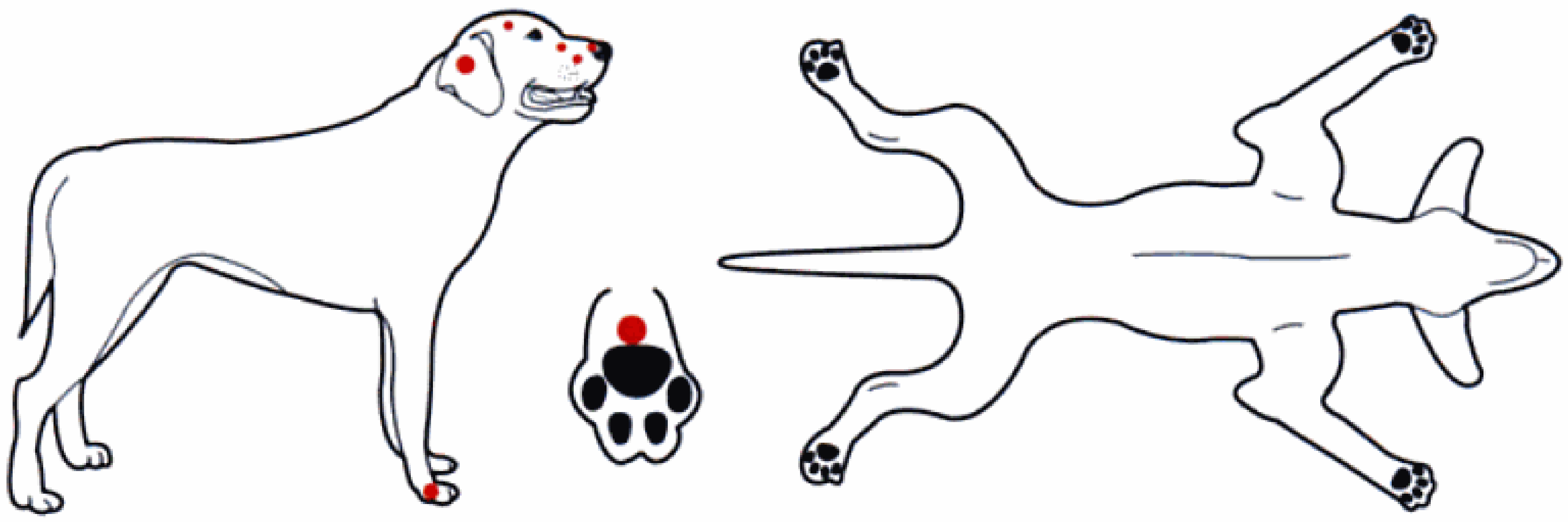
Aetiology: Idiopathic infiltration of the skin with macrophages and neutrophils.

Predominant lesions:

Nodules and plaques.

Lesion distribution:

Head, pinnae paws.



Appropriate diagnostic tests:

Cytology
Biopsy
Tests to rule out infectious causes (see Chapter 9)

Cutaneous, systemic and malignant histiocytosis

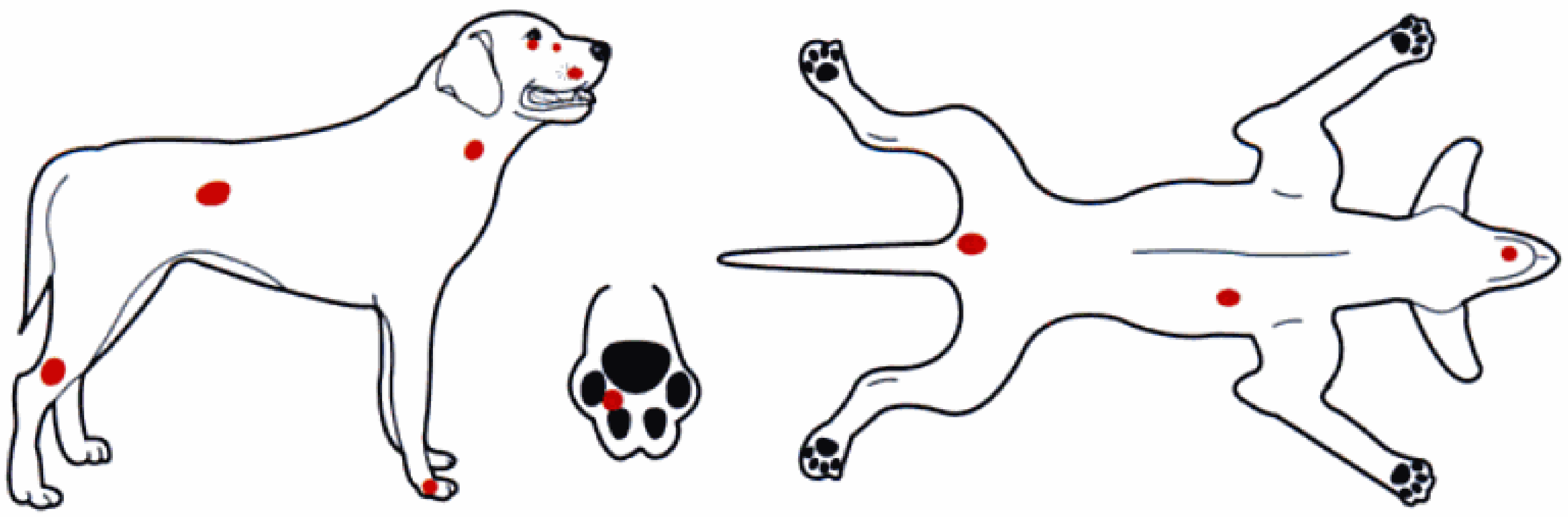
Aetiology: Idiopathic infiltration of the skin (and sometimes internal organs) with histiocytes. Cutaneous histiocytosis can occur in any breed and is similar to sterile granuloma syndrome (described above). Systemic and malignant histiocytosis occur most commonly in Bernese mountain dogs. In addition to cutaneous lesions, these dogs have systemic involvement.

Predominant lesions:

Multiple, firm, nodules and plaques (may be ulcerated in systemic forms)
Systemic signs can include anorexia, weight loss, respiratory distress, ocular abnormalities, lymphadenopathy, hepatomegaly and pancytopaenia.

Lesion distribution:

Anywhere on the body.



Appropriate diagnostic tests:

Cytology
Biopsy
Haematology and Biochemistry

Plasma cell pododermatitis

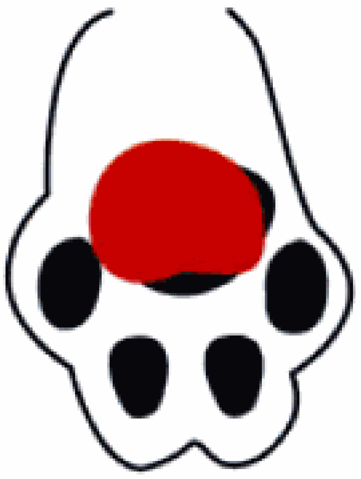
Aetiology: An idiopathic infiltration of the footpad with plasma cells, seen only in cats.

Predominant lesions:

Soft, smooth swelling that may ulcerate.

Lesion distribution:

Footpads.



Appropriate diagnostic tests:

Cytology
Biopsy

Cysts

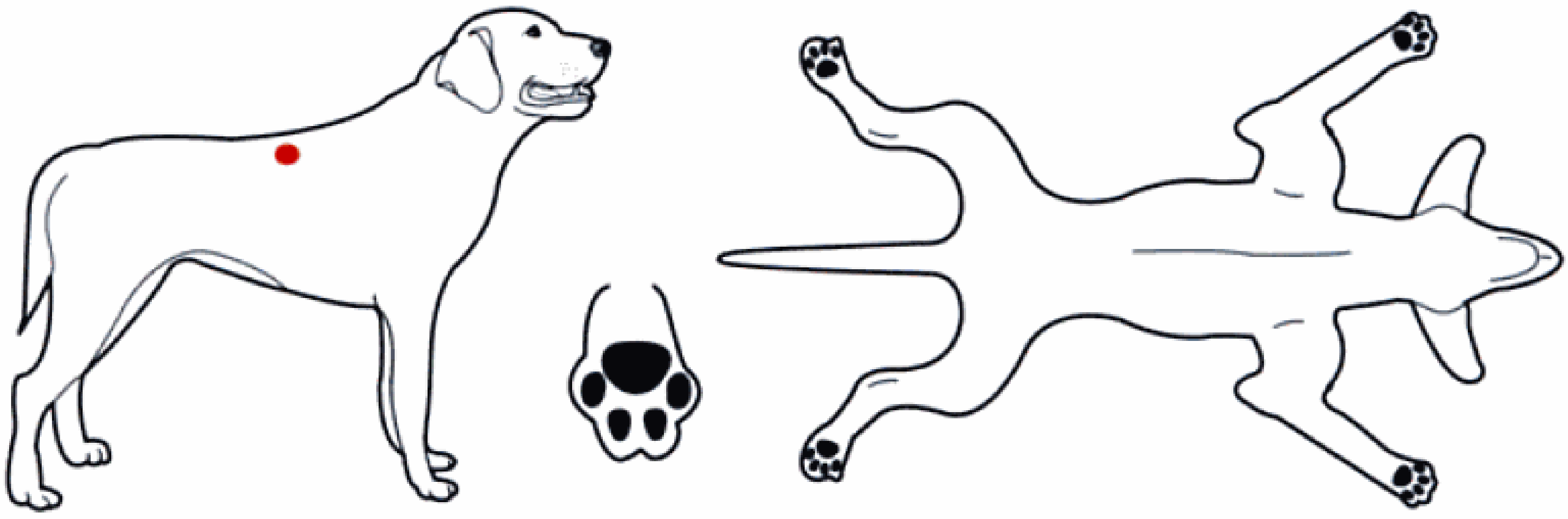
Aetiology: Non-neoplastic, epithelial lined sacs containing a variety of skin components depending on the epithelial origin. Examples include follicular cysts, epidermal inclusion cysts, dermoid cysts, sweat gland cysts and sebaceous cysts.

Predominant lesions:

Single or multiple firm swellings.

Lesion distribution:

Anywhere on the body.



Appropriate diagnostic tests:

Cytology
Biopsy

Lipoma

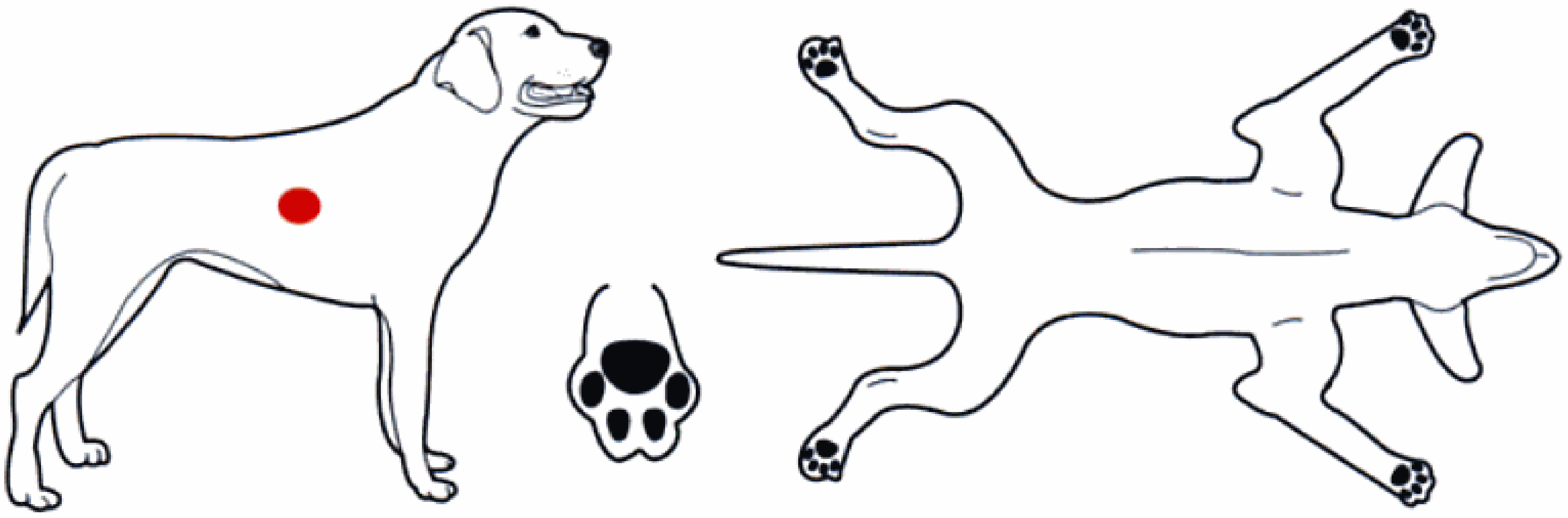
Aetiology: A very common skin tumour in dogs caused by the neoplastic proliferation of fat cells (lipocytes, adipocytes). Uncommon in cats.

Predominant lesions:

Well circumscribed, soft to flabby, subcutaneous mass.

Lesion distribution:

Usually on the trunk or proximal limbs.



Appropriate diagnostic tests:

Cytology
Biopsy

Sebaceous gland hyperplasia/adenoma

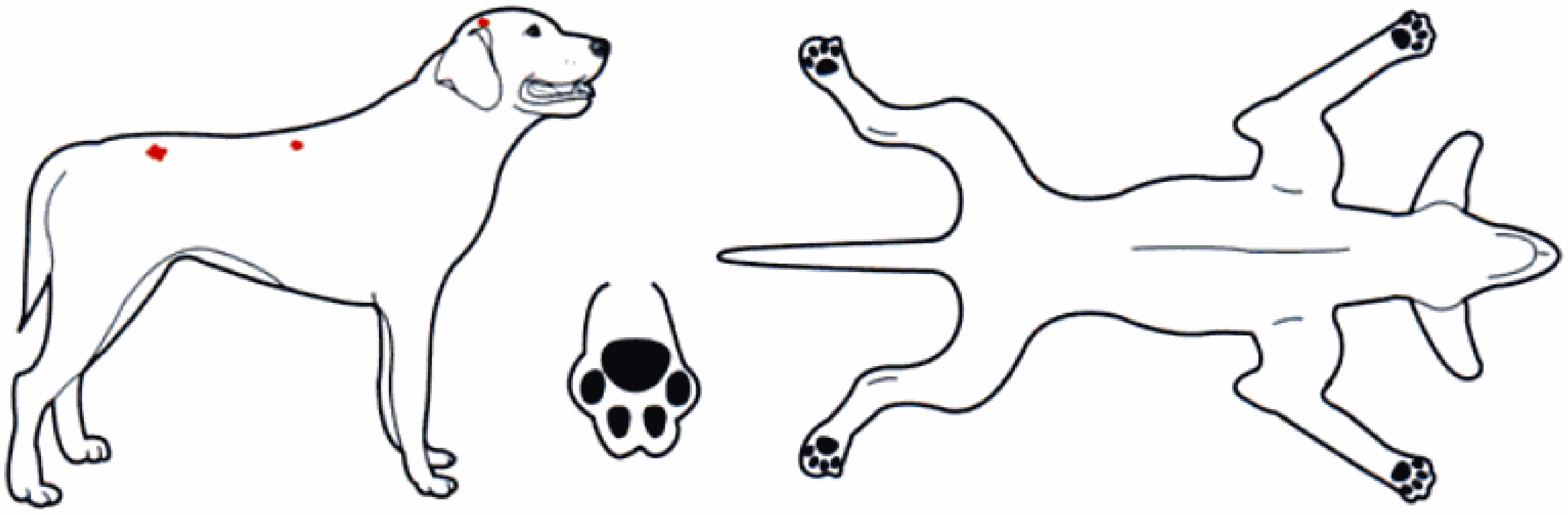
Aetiology: A very common skin tumour in dogs caused by hyperplastic or neoplastic proliferation of sebaceous gland cells (sebocytes). Uncommon in cats.

Predominant lesions:

Single to multiple, wart-like pinkish tumours that look like miniature brains or cauliflowers.

Lesion distribution:

Head, eyelids and trunk.



Appropriate diagnostic tests:

Cytology
Biopsy

Tail gland hyperplasia

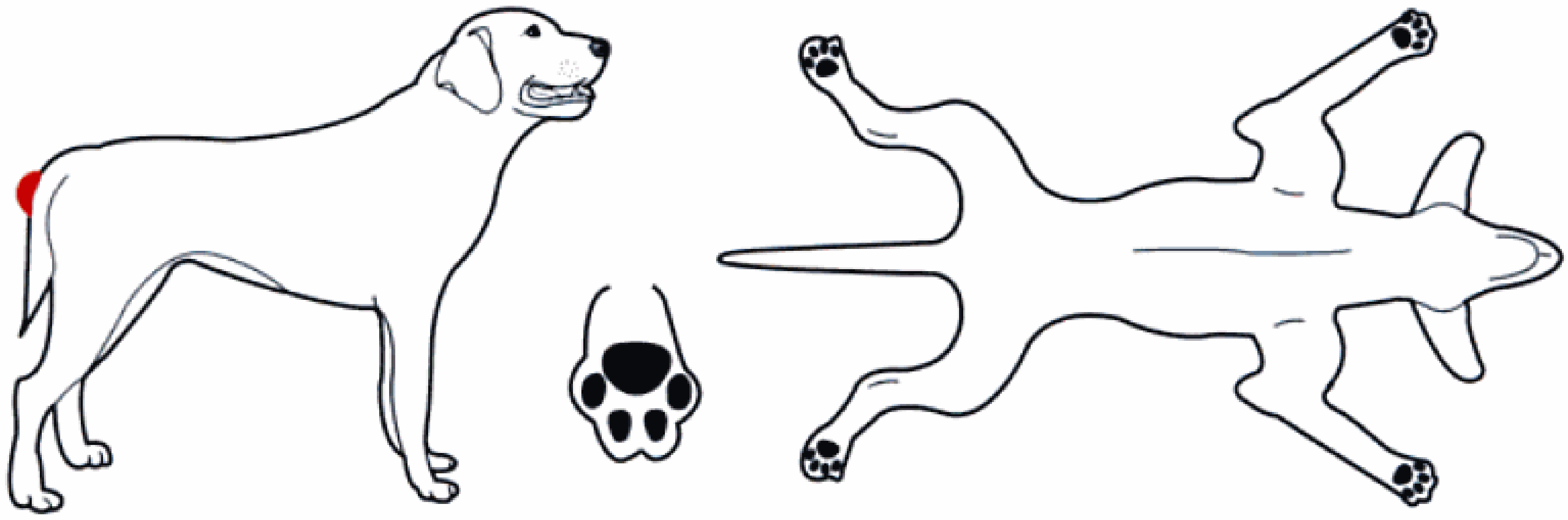
Aetiology: A common problem of dogs caused by hyperplastic proliferation of sebaceous gland cells of the tail. May be associated with testicular tumours but is often idiopathic.

Predominant lesions:

Swelling.

Lesion distribution:

Dorsal tail.



Appropriate diagnostic tests:

Rule out testicular neoplasia

Mast cell tumour

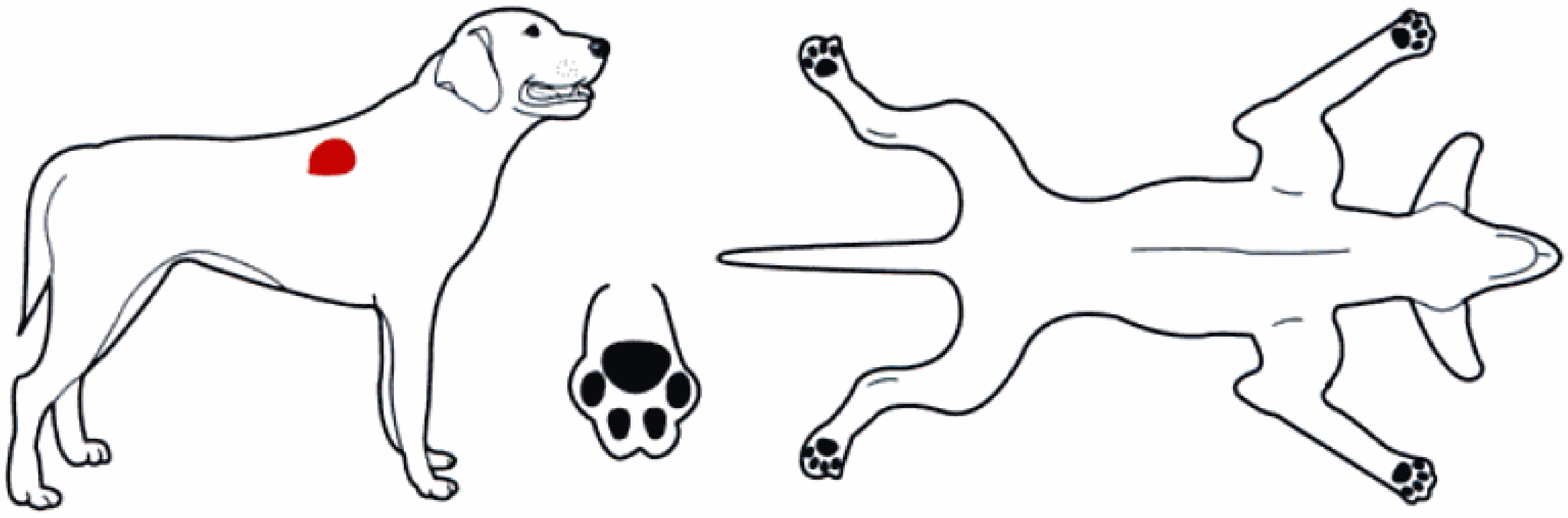
Aetiology: A common tumour caused by neoplastic proliferation of mast cells.

Predominant lesions:

Single to multiple nodules or tumours; areas of diffuse swelling.

Lesion distribution:

Anywhere on the body.



Appropriate diagnostic tests:

Cytology
Biopsy

Papilloma (warts)

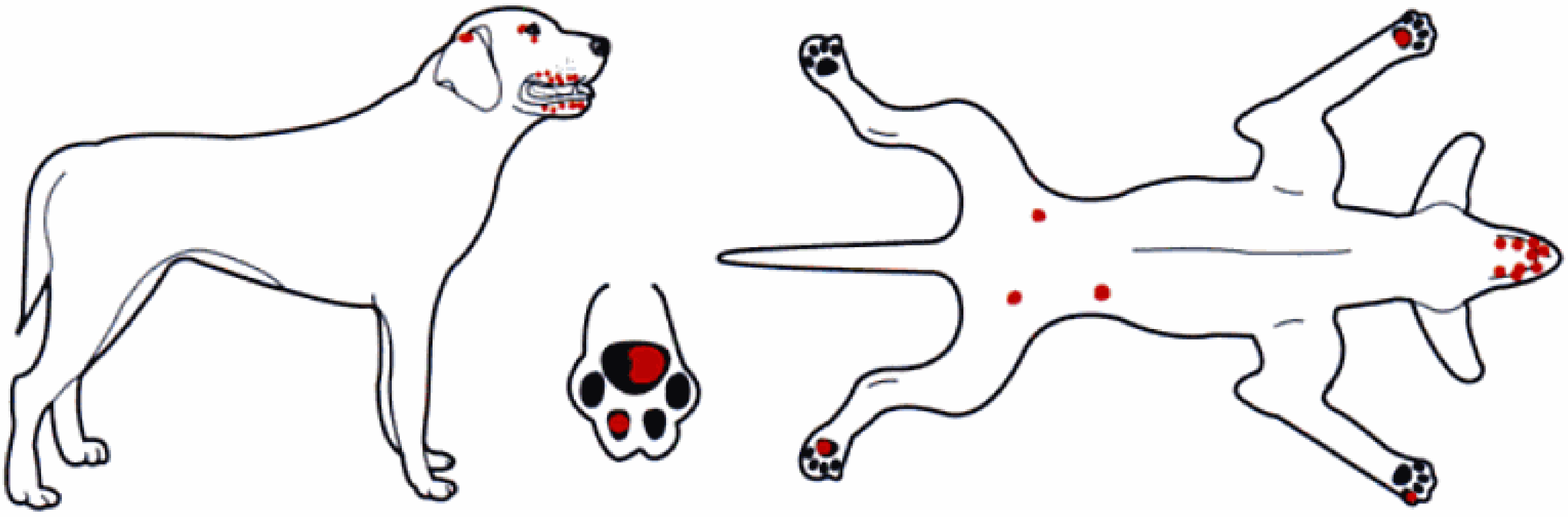
Aetiology: Neoplastic proliferation of epidermal cells usually induced by a papilloma virus.

Predominant lesions:

Single to multiple, cauliflower-like tumours.

Lesion distribution:

Oral papillomatosis affects the oral cavity, lips, eyelids. Cutaneous papillomas may occur anywhere on the body.



Appropriate diagnostic tests:

Cytology
Biopsy

Squamous cell carcinoma

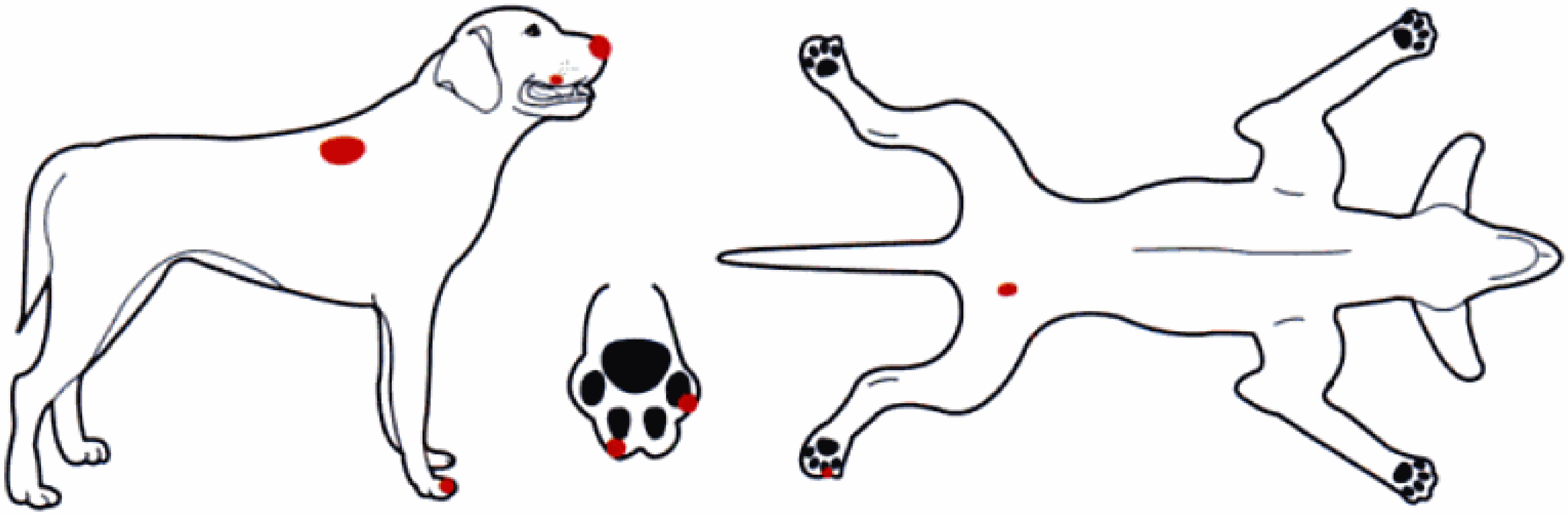
Aetiology: Neoplastic proliferation of keratinocytes that may be precipitated by excessive exposure to UV light.

Predominant lesions:

Proliferative or ulcerative tumours.

Lesion distribution:

Dogs – anywhere on the body, including the clawbeds.
Cats – Pinnae, nasal planum, eyelids (also see Chapter 8).



Appropriate diagnostic tests:

Cytology
Biopsy

Basal cell tumour

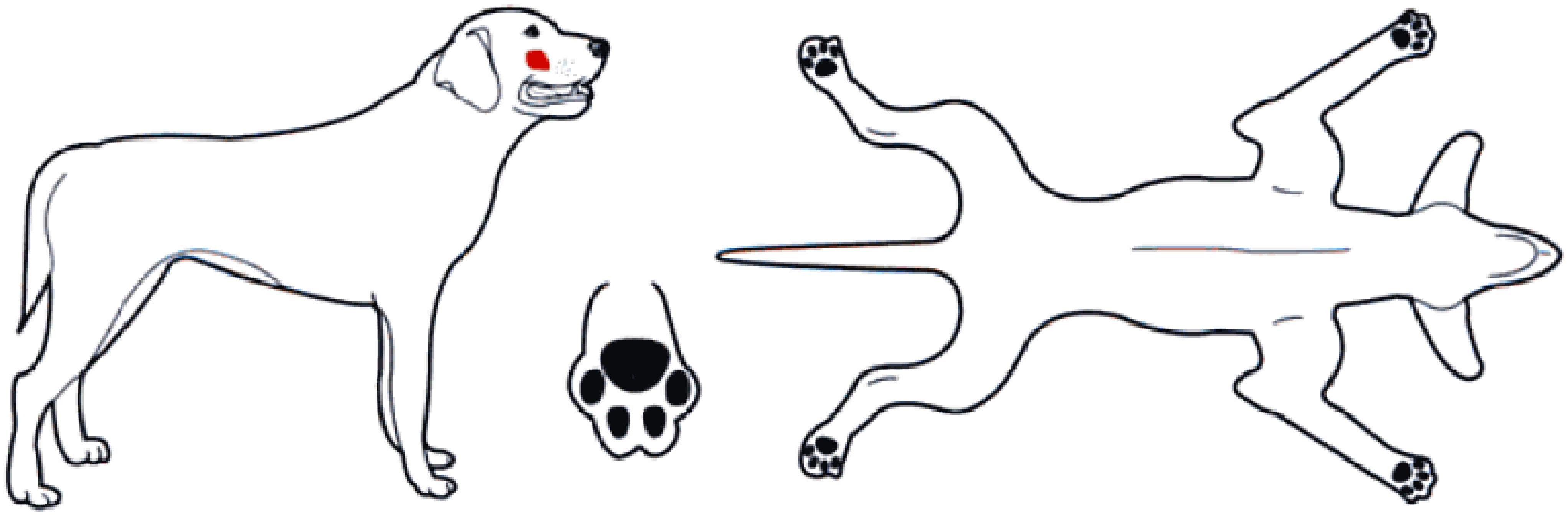
Aetiology: Neoplastic proliferation of basal keratinocytes.

Predominant lesions:

Tumours that may be ulcerated or pigmented.

Lesion distribution:

Head, neck, limbs.



Appropriate diagnostic tests:

Cytology
Biopsy

Perianal gland tumours

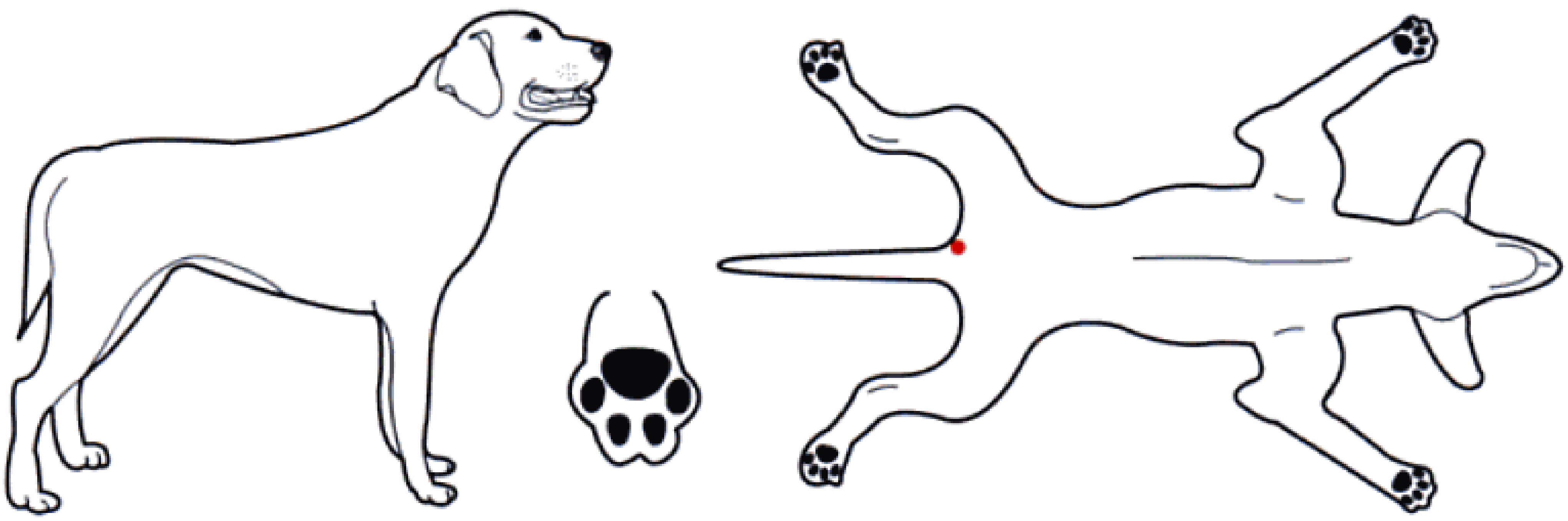
Aetiology: A common tumour of the dog caused by neoplastic proliferation of circumanal (hepatoid) glands or anal sac glands. In male dogs, proliferation of the hepatoid glands is under the influence of androgens and oestrogens from the testes.

Predominant lesions:

Single to multiple tumours.

Lesion distribution:

Adjacent to the anus.



Appropriate diagnostic tests:

Cytology
Biopsy
Haematology and Biochemistry

Hair follicle tumours

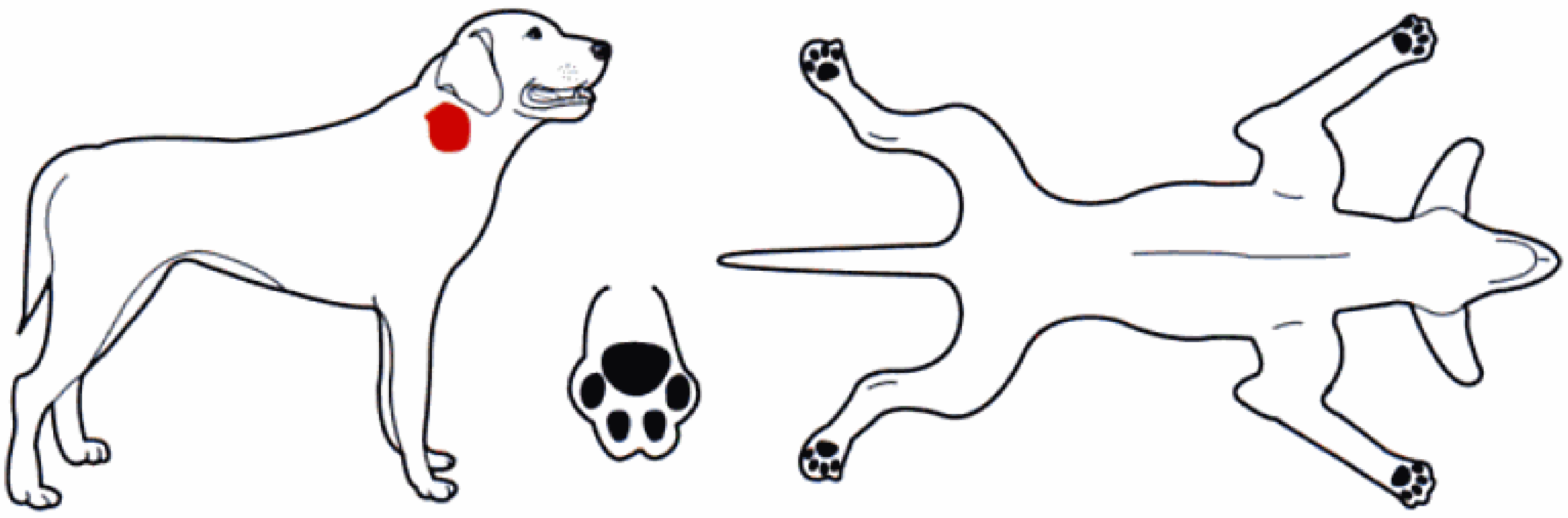
Aetiology: Neoplastic proliferation of epithelial structures that make up the hair follicle.

Predominant lesions:

Tumours.

Lesion distribution:

Anywhere on the body.



Appropriate diagnostic tests:

Cytology
Biopsy

Sweat gland tumours

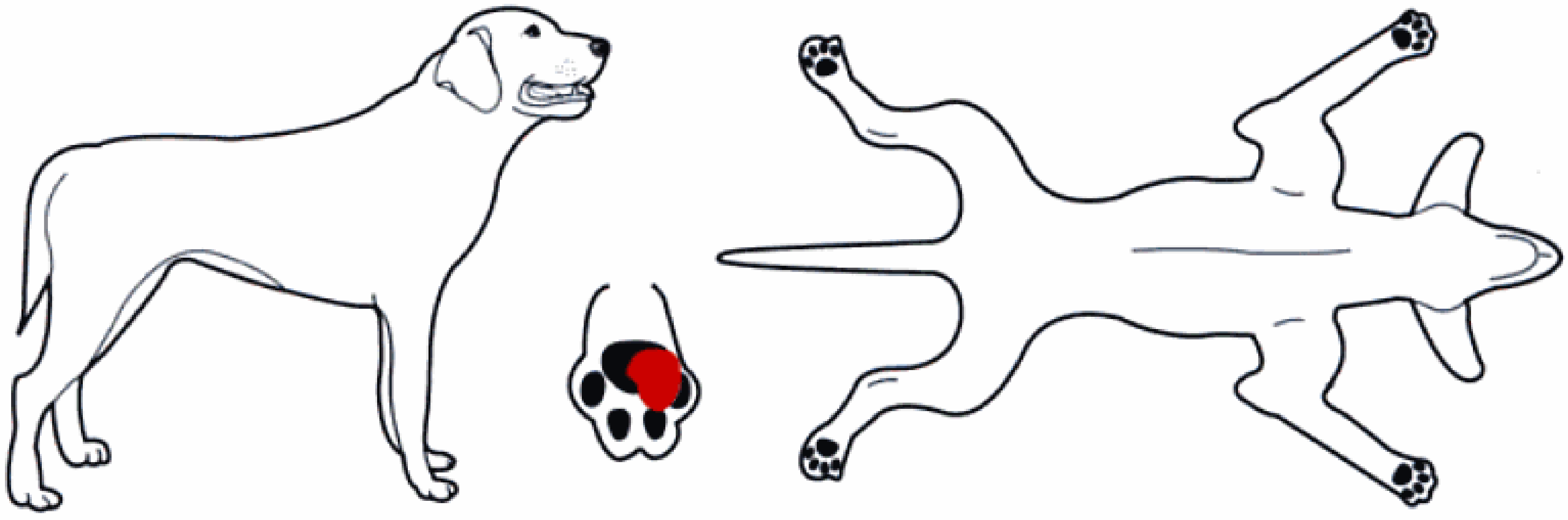
Aetiology: Neoplastic proliferation of sweat gland epithelium.

Predominant lesions:

Tumours.

Lesion distribution:

Head, neck, trunk, limbs, footpads.



Appropriate diagnostic tests:

Cytology
Biopsy

Fibroblast tumours (fibroma/fibrosarcoma)

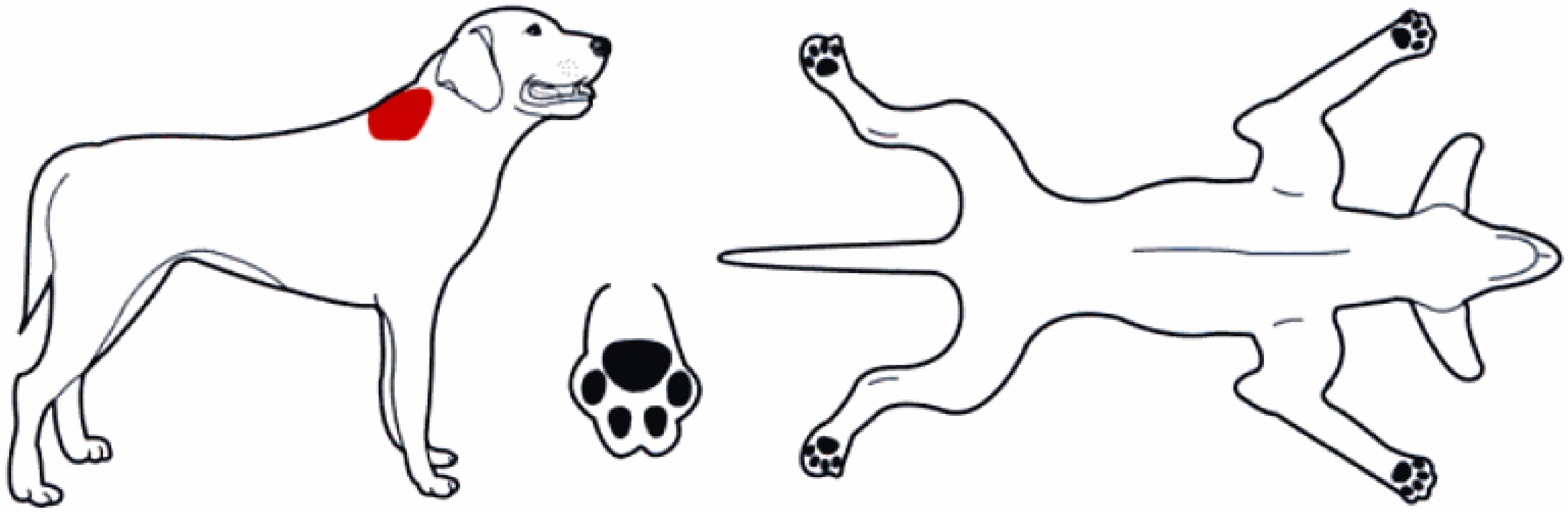
Aetiology: Neoplastic proliferation of fibroblasts. May occur spontaneously in dogs and cats, but in cats it may also occur secondary to Feline Sarcoma virus or at the site of previous vaccinations.

Predominant lesions:

Single to multiple tumours.

Lesion distribution:

Anywhere on the body.



Appropriate diagnostic tests:

Cytology
Biopsy

Haemangiopericytoma

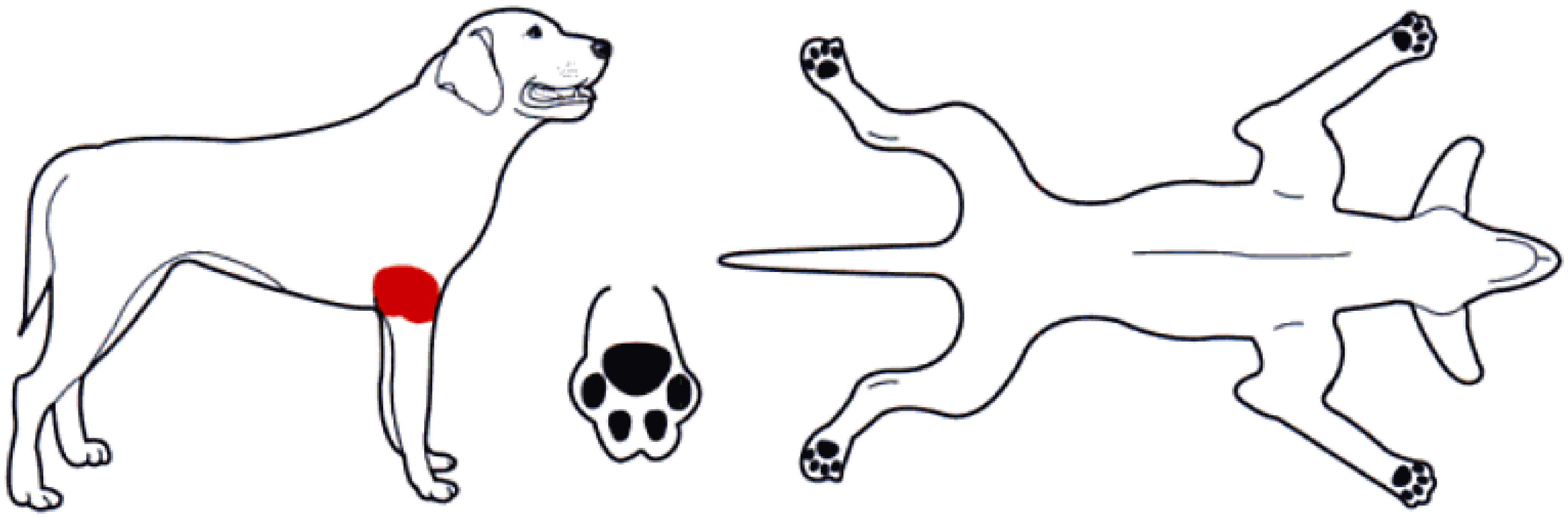
Aetiology: A common tumour of dogs caused by neoplastic proliferation of vascular pericytes.

Predominant lesions:

Tumours.

Lesion distribution:

On the limbs, especially the stifle and elbow.



Appropriate diagnostic tests:

Cytology
Biopsy

Neural tumours (schwannoma and tail dock neuroma)

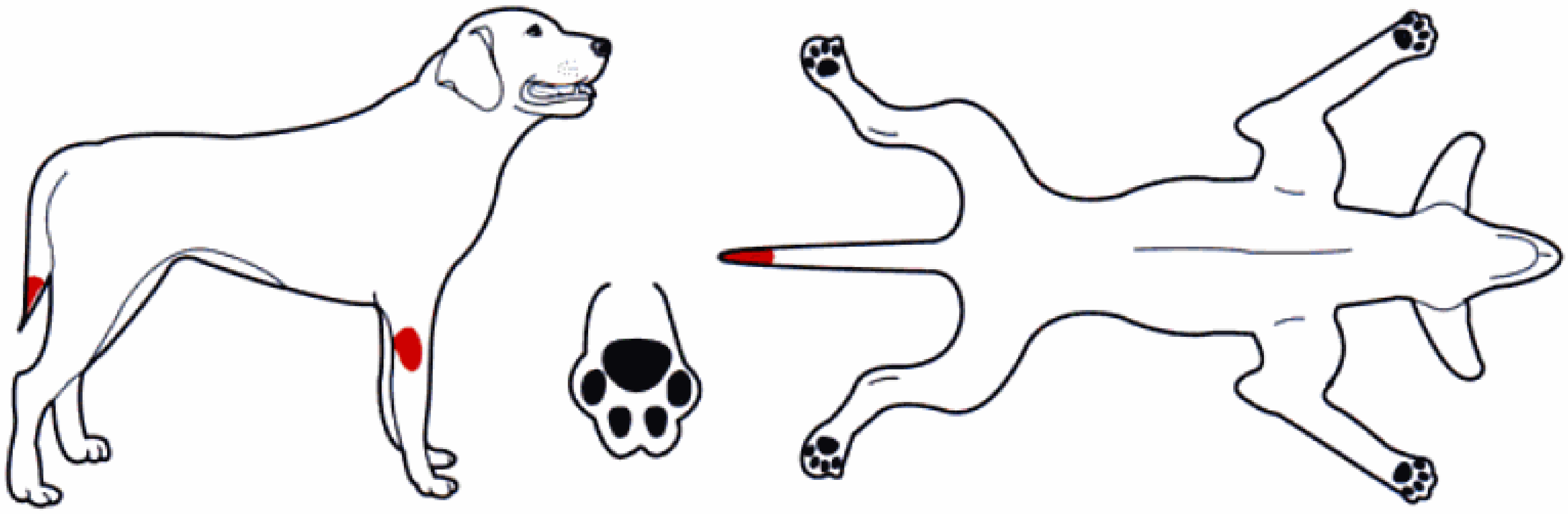
Aetiology: A schwannoma is a neoplastic proliferation of cutaneous Schwann cells. A tail dock neuroma is a disorganised proliferation of nerve axons following tail docking.

Predominant lesions:

Tumours.

Lesion distribution:

Schwannoma – limbs, head, tail and neck.
Tail dock neuroma – tail stump.



Appropriate diagnostic tests:

Cytology
Biopsy

Vascular tumours (haemangioma, haemangiosarcoma)

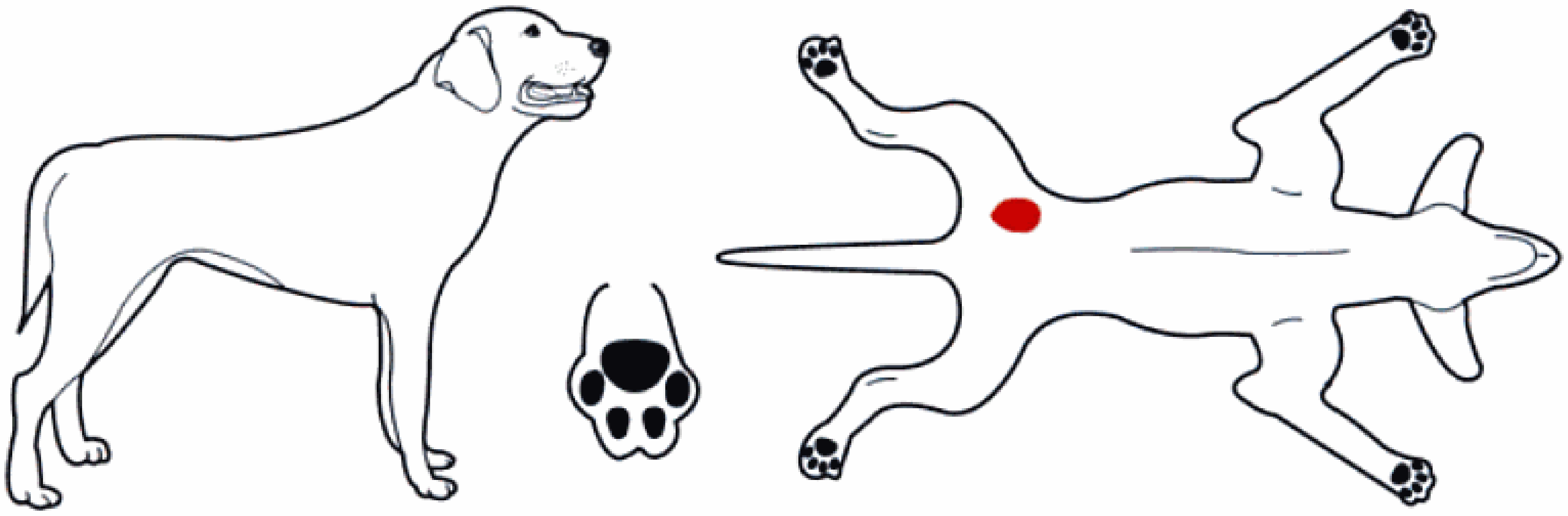
Aetiology: Neoplastic proliferation of blood vessel endothelial cells.

Predominant lesions:

Bluish to reddish black tumours (may appear as a blood-filled swelling – see earlier).

Lesion distribution:

Anywhere on the body.



Appropriate diagnostic tests:

Cytology
Biopsy

Lymphoid tumours (lymphoma)

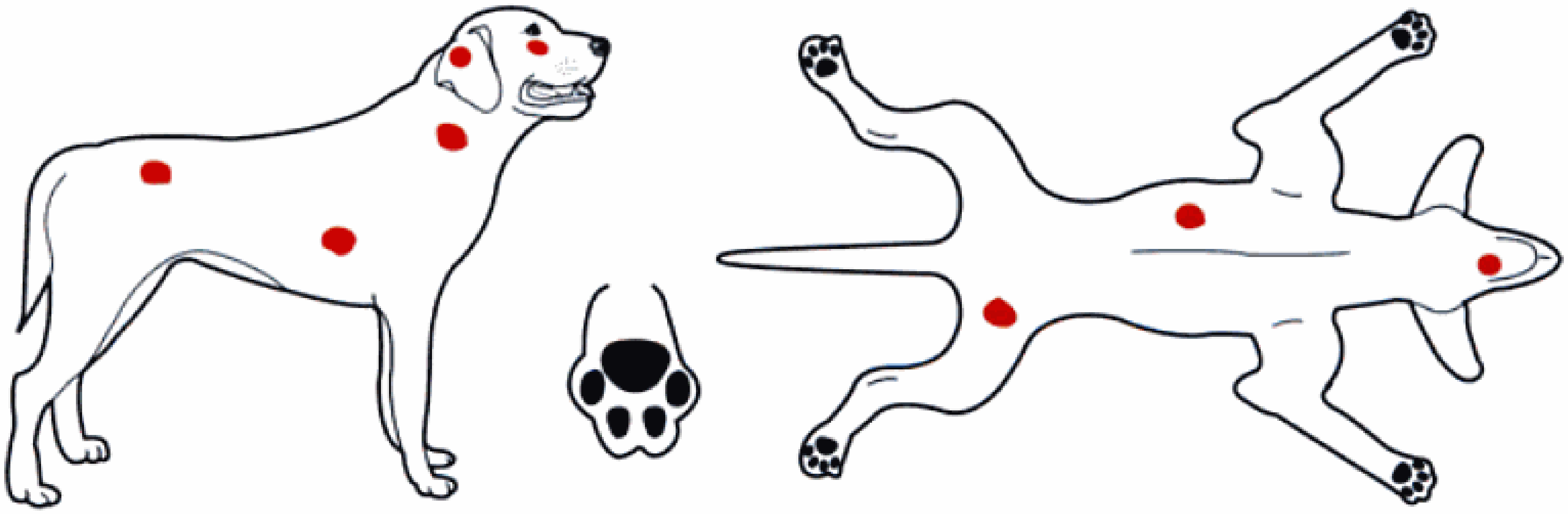
Aetiology: Neoplastic proliferation of lymphocytes. Cutaneous lymphoma can be epitheliotropic (described in Chapter 4) or non-epitheliotropic.

Predominant lesions:

Scaling, erythroderma, alopecia, exfoliation, hypopigmentation, plaques, nodules, ulcers, tumours.

Lesion distribution:

Multifocal or generalised.



Appropriate diagnostic tests:

Cytology
Biopsy
Haematology and Biochemistry

Histiocytoma

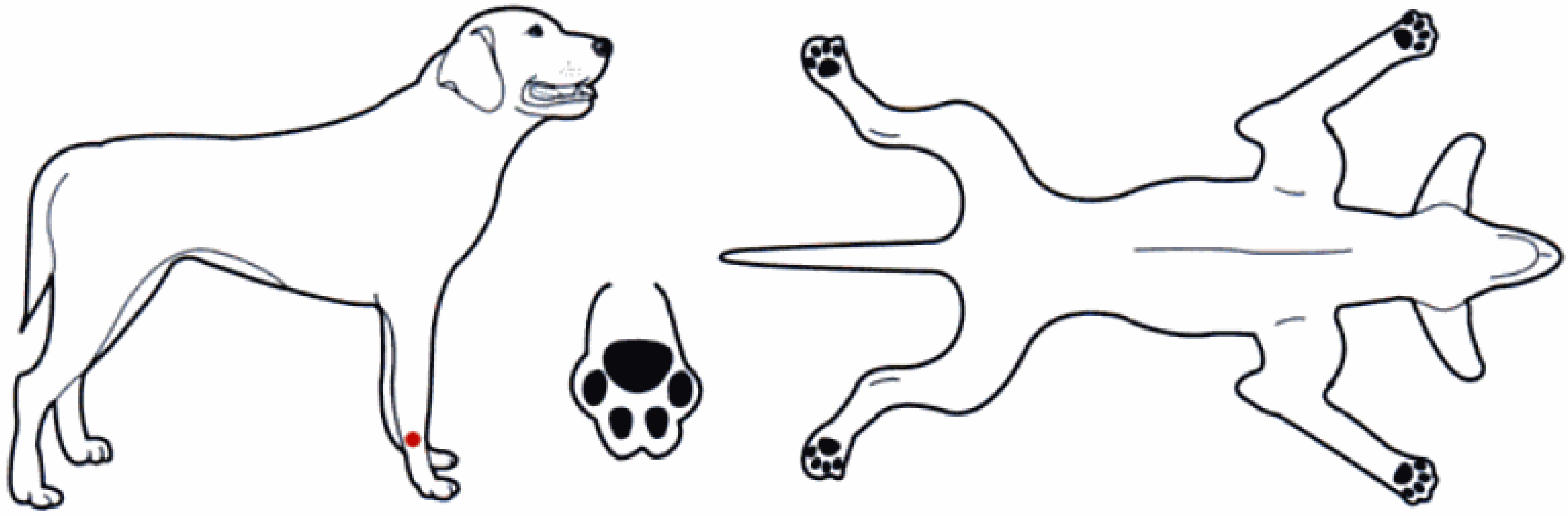
Aetiology: A common tumour of dogs caused by a neoplastic proliferation of epidermal Langerhans cells. Usually seen in young dogs and the majority resolve spontaneously within 3 months.

Predominant lesions:

Single, rapidly growing, dome-shaped tumours that may be ulcerated.

Lesion distribution:

Head, pinnae, limbs.



Appropriate diagnostic tests:

Cytology
Clinical diagnosis
Biopsy

Plasma cell tumour

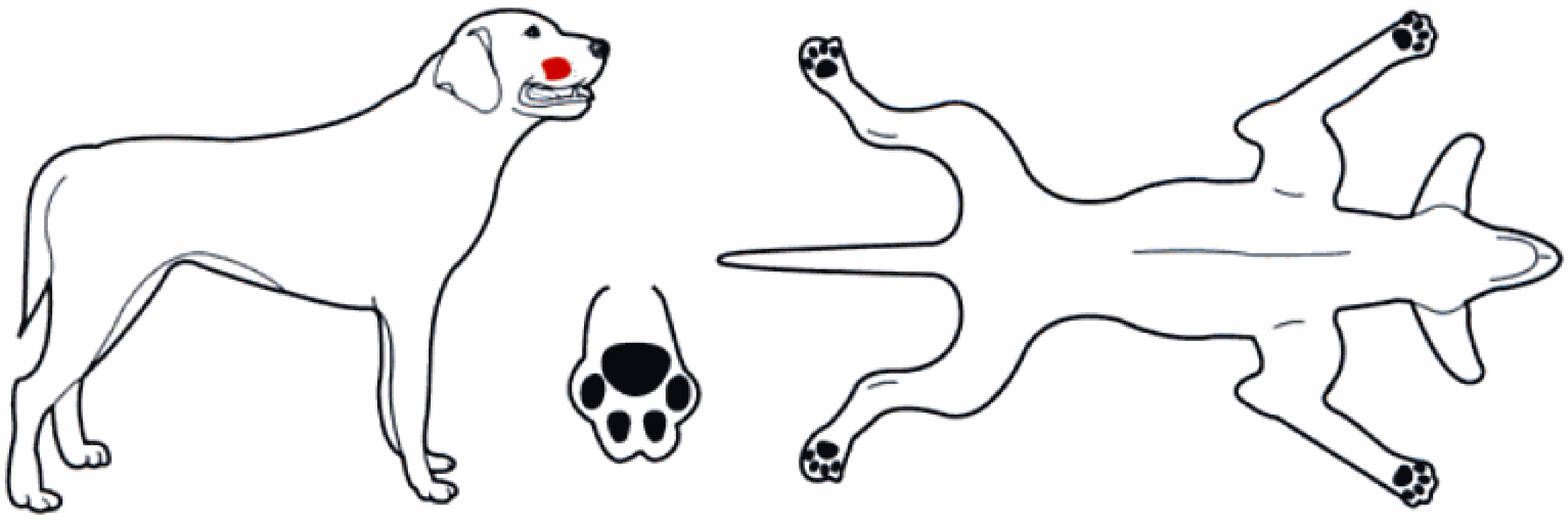
Aetiology: Neoplastic proliferation of plasma cells seen commonly in dogs but rarely in cats.

Predominant lesions:

Nodules, tumours.

Lesion distribution:

Digits, lips, chin, ear canal, but can be anywhere.



Appropriate diagnostic tests:

Cytology
Biopsy

Melanoma

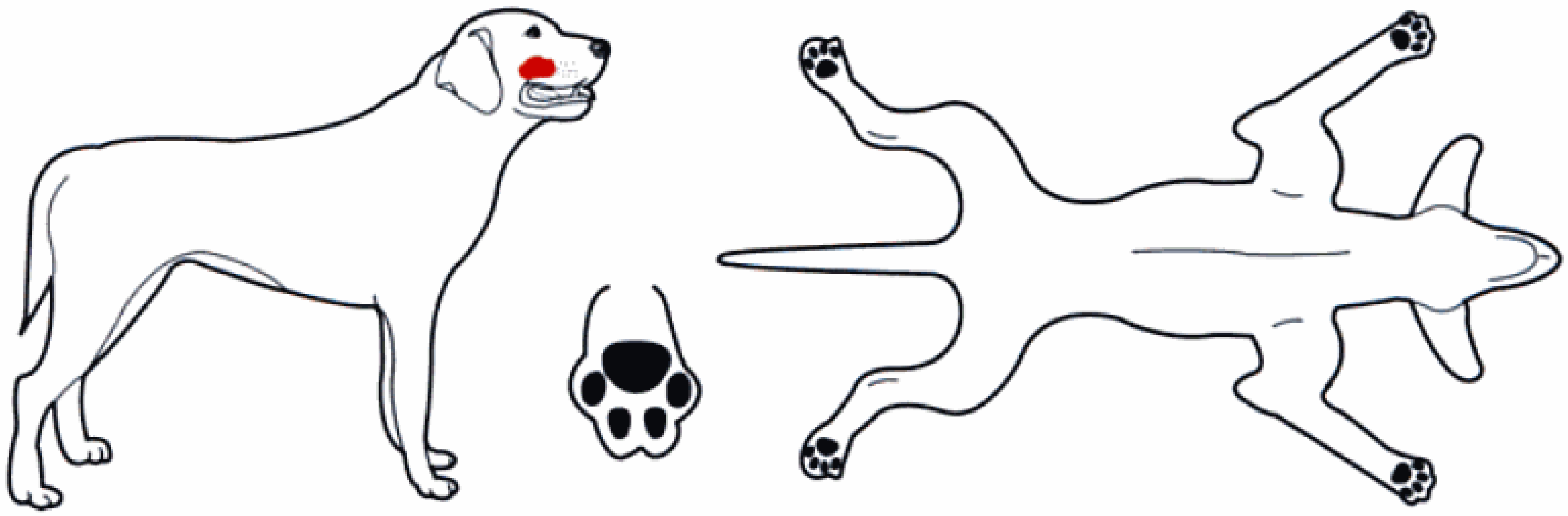
Aetiology: Neoplastic proliferation of melanocytes.

Predominant lesions:

Pigmented tumours.

Lesion distribution:

Head, neck, eyelids, muzzle, trunk, paws.



Appropriate diagnostic tests:

Cytology
Biopsy

Calcinosis cutis

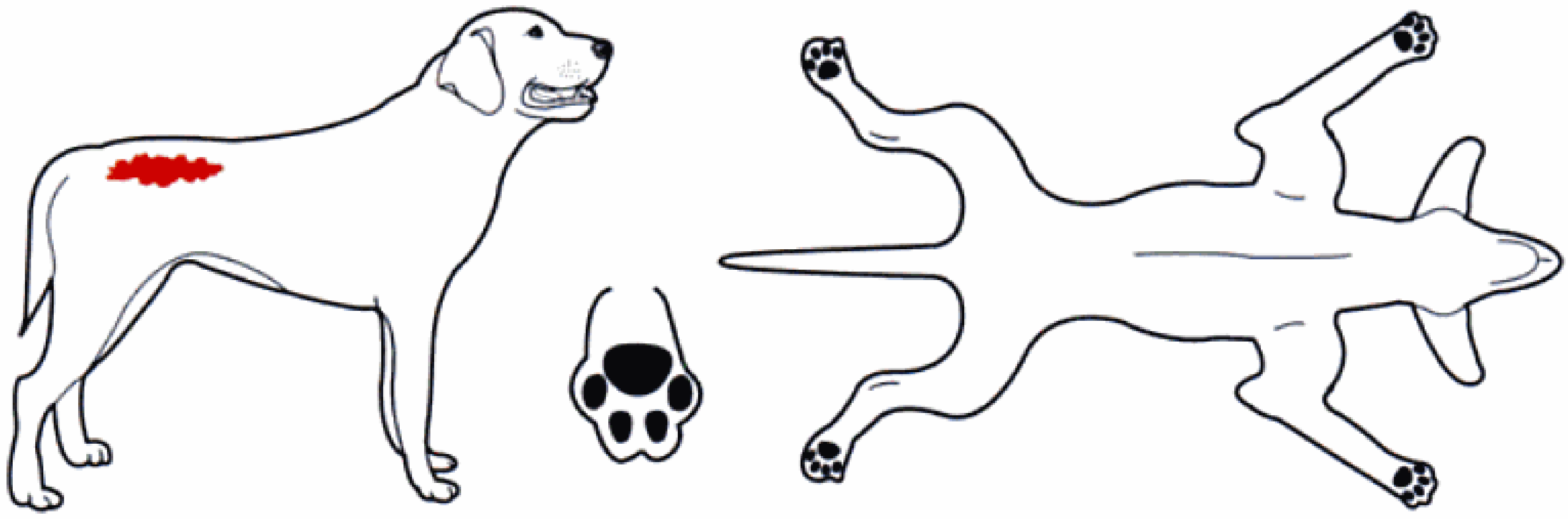
Aetiology: Deposition of calcium in the skin associated with hyperadrenocorticism.

Predominant lesions:

Firm, gritty, whitish plaques and nodules that may become ulcerated.

Lesion distribution:

Dorsum, axillae, inguinal region.



Appropriate diagnostic tests:

Clinical diagnosis
Biopsy
Tests for hyperadrenocorticism

Calcinosis circumscripta

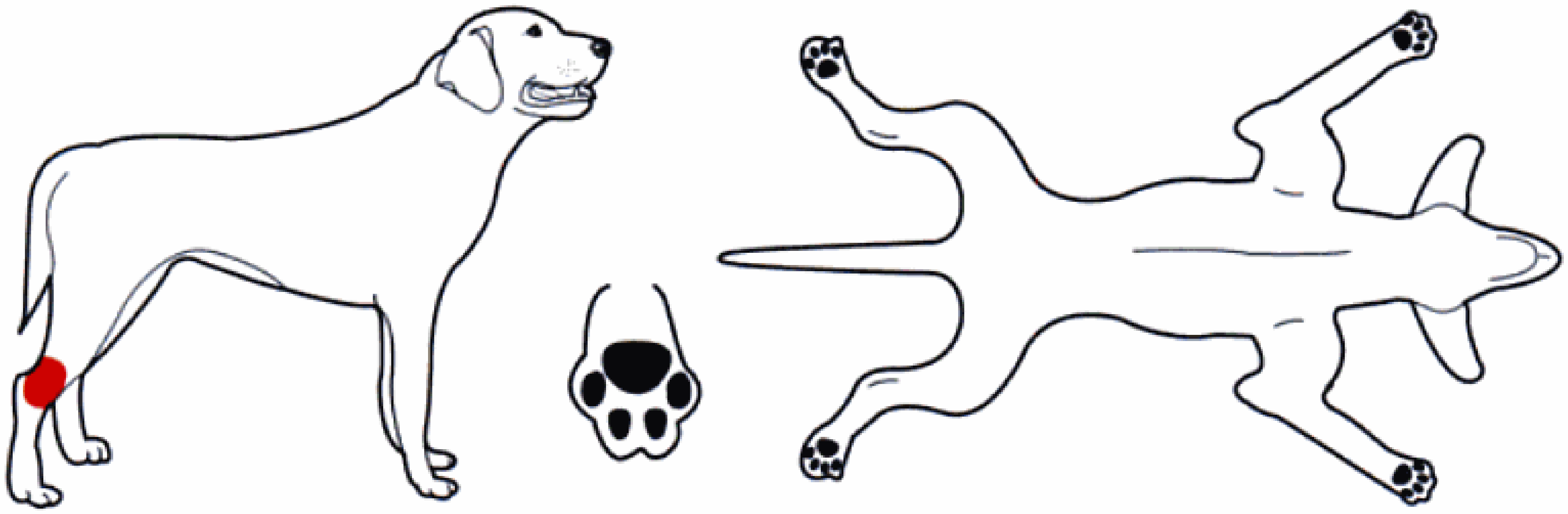
Aetiology: Deposition of calcium in the skin over pressure points or at sites of trauma. Most commonly occurs in large dogs, especially German shepherd dogs.

Predominant lesions:

Solitary lumps.

Lesion distribution:

Hock, elbow, tongue.



Appropriate diagnostic tests:

Radiography
Biopsy

Mucinosis

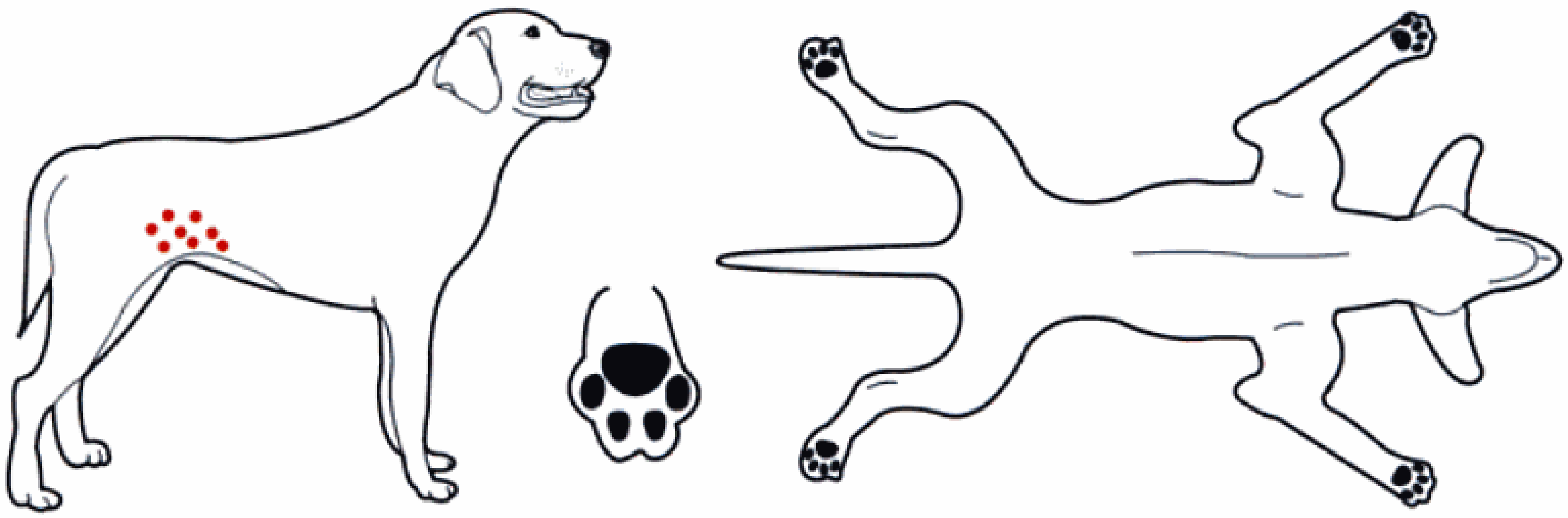
Aetiology: Accumulation of mucin within the skin. Occurs commonly in the Shar Pei.

Predominant lesions:

Single to multiple, soft, rubbery swellings.

Lesion distribution:

Trunk.



Appropriate diagnostic tests:

Cytology
Biopsy

Naevus

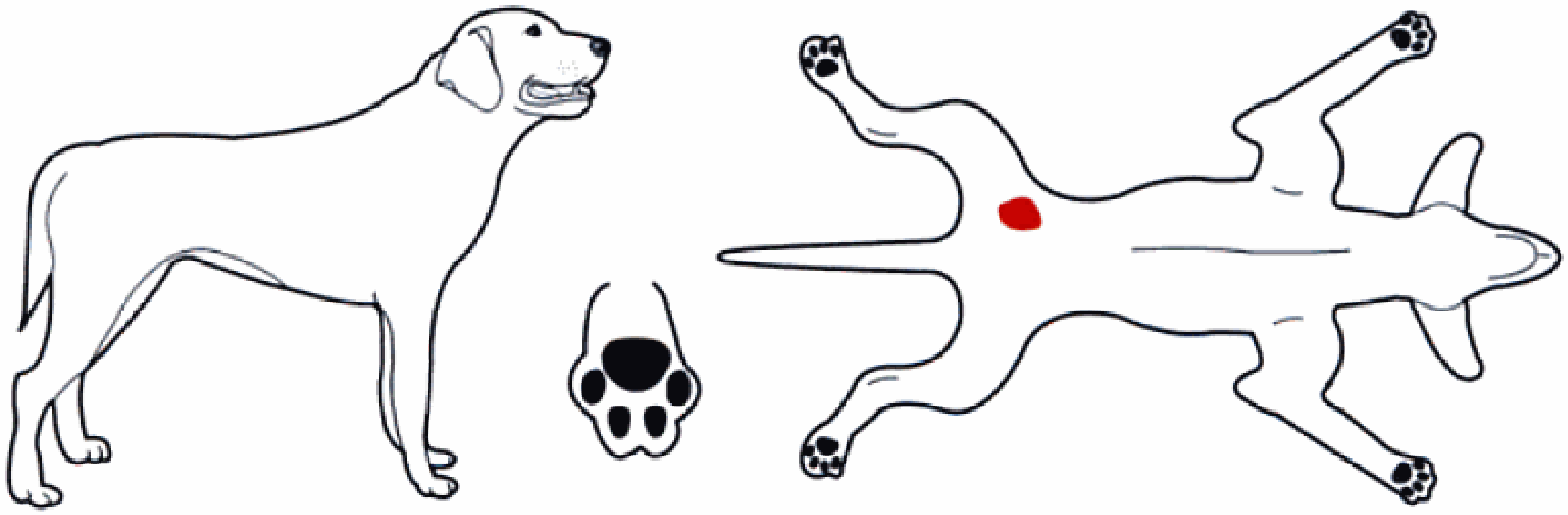
Aetiology: Developmental abnormality in the skin caused by hyperplasia of various skin components (collagen, vascular tissue, glands, epidermis or melanocytes).

Predominant lesions:

Lumps.

Lesion distribution:

Anywhere.



Appropriate diagnostic tests:

Biopsy

Dermatofibrosis

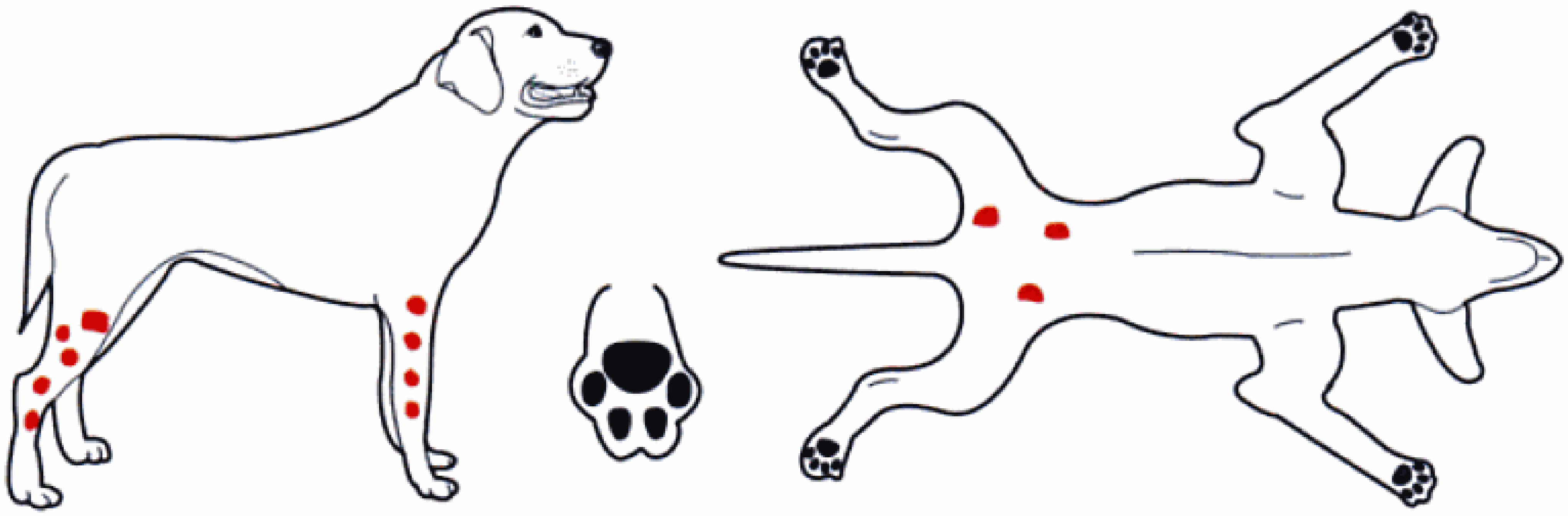
Aetiology: Multiple collagenous naevi seen in association with renal cystadenocarcinomas or leiomyomas. Usually seen in German shepherd dogs but rarely in other breeds.

Predominant lesions:

Multiple nodules and swellings.

Lesion distribution:

Limbs, head, neck, ventrum.



Appropriate diagnostic tests:

Biopsy
Haematology
Biochemistry
Abdominal ultrasound

General diagnostic approach to lumps and swellings

As with other dermatological problems, the investigation of a lump or swelling should begin with a thorough history and physical examination. This may reveal evidence of trauma or bite wounds that could have caused haematomas or abscesses. It is also important to determine how rapidly a lump appeared, how long it has been there and whether it is increasing in size. A full dermatological examination should be performed to check for the presence of other lumps elsewhere. It is not uncommon for animals to be presented for examination of a single lump when multiple lumps can be found. The lump itself should then be carefully examined. Visual inspection determines the appearance, size, shape, and the presence of any discharging sinuses or ulceration. Palpation determines the consistency, firmness, depth and attachment to underlying tissues.

In some cases, the history and clinical examination of a lump or swelling can be very suggestive of the diagnosis. Examples where this is possible include aural haematomas, urticaria, angioedema, seromas, cat bite abscesses, calcinosis cutis, sebaceous adenomas, perianal adenomas and histiocytomas. In most other cases, the clinical appearance is not pathognomonic and accurate diagnosis and prognosis relies on diagnostic tests. It is not possible to determine what a lump is, or if it is benign or malignant, by observation and palpation. It is therefore not appropriate for a clinician to feel a lump and tell a client to keep an eye on it to see if it grows.

Unless a specific diagnosis has been made based on the history and physical examination alone, the clinician should perform a fine needle aspirate. The initial goal of this technique is to determine the predominant cellular content (see Table 11.1). This may provide a definitive diagnosis, a suspected diagnosis or no diagnosis. Fine needle aspiration cytology can provide a definitive diagnosis for abscesses, cysts, lipomas, sebaceous gland adenomas and mast cell tumours. Fortunately, these are the most common causes of cutaneous lumps seen in general practice. Fine needle aspirates may also enable a tumour type to be categorised as epithelial, spindle, round cell or melanocytic. Although this is useful information, a biopsy of the mass or the whole mass should be submitted for histopathological analysis to allow more specific differentiation.

In some cases, fine needle aspiration may be totally unrewarding. In these cases, it is essential to perform biopsy and histopathological analysis in order to make a specific diagnosis. Details on performing fine needle aspirates and skin biopsies are described in Chapter 13.

12

Otitis

Clinical features and differential diagnosis

Otitis is inflammation of the ear canal. Otitis may be restricted to the vertical and horizontal ear canals (otitis externa) or it may affect the middle ear cavity (otitis media). This usually occurs when infection spreads through the tympanic membrane into the tympanic bullae. Otitis interna refers to inflammation that has reached the cochlea or semicircular canals.

Otitis externa is a very common presenting problem in dogs but less common in cats. The usual clinical signs are aural pruritus, aural pain, head shaking, malodour and the presence of a visible discharge. The clinical signs of otitis media are identical to those of otitis externa but they tend to be more persistent and recurrent. In addition, hearing may be impaired due to damage of the tympanic membrane and ossicles. The clinical signs of otitis interna are rare and include deafness and vestibular disease (head tilt, nystagmus, ataxia).

In the majority of cases of otitis externa and media, the visible discharge is due to a bacterial or yeast infection. Bacteria commonly associated with otitis include *Staphylococcus intermedius, Streptococcus canis, Proteus* spp., *Escherichia coli, Klebsiella* spp. and *Pseudomonas aeruginosa. Malassezia pachydermatis* is the most common yeast that contributes to otitis. Bacterial infections tend to produce a yellow or green purulent discharge whereas yeast infections tend to produce a greasy, brownish, waxy discharge.

As with other types of skin infection, ear infections usually occur secondary to an underlying cause. Hence, when presented with a case of otitis, the clinician should always attempt to determine what has lead to the infection as well as treating the infection itself. Table 12.1 show the various factors and diseases that are known to lead to otitis. In this table, the underlying causes are divided into predisposing factors, primary causes and perpetuating factors. Predisposing factors make an animal more prone to the development of otitis but do not necessarily cause it. Primary causes are conditions in which otitis is a common manifestation. Perpetuating factors are reasons why a case of otitis becomes recurrent or resistant to treatment.

General diagnostic approach to otitis

As with other dermatological problems, a full history and physical examination is required when investigating cases of otitis. This is to ensure that potential underlying causes are not missed at the outset. In addition, an otoscopic examination must be performed. The aim of the otoscopic examination is to detect foreign bodies or ear mites, to observe the condition of the vertical and horizontal canals, to check the appearance and integrity of the tympanic membrane, and to characterise the type of exudate that is present. If the condition is unilateral, clinicians should always examine the good ear first. This prevents the

Table 12.1 Underlying causes of otitis

Type of factor	Specific condition
Predisposing factors	**Pendulous pinnae (D)**
	Narrow ear canals (D)
	Hairy ear canals (D)
	Excessive wax production (D)
	Swimming (D)
Primary causes	**Foreign bodies (esp. grass seeds) (D)**
	***Otodectes cynotis* infestation (ear mites) (D and C)**
	Otic demodicosis (D and C)
	Atopic dermatitis (D)
	Food allergy or intolerance (D and C)
	Adverse reaction to ear medication (D and C)
	Hypothyroidism (D)
	Seborrhoeic conditions (D)
	Ear canal tumours (D and C)
	Ceruminous gland hyperplasia (D and C)
	Lupus (D and C)
	Pemphigus (D and C)
	Juvenile cellulitis (D)
Perpetuating factors	**Otitis media (D and C)**
	Resistant bacterial infections (esp. Pseudomonas) (D)
	Progressive narrowing of the ear canals (D)
	Ear canal fibrosis (D)
	Ear canal calcification (D)
	Bulla osteomyelitis (D)

Those in bold would be most common in general practice. D = those diseases seen in dogs, C = those diseases seen in cats.

spread of infection from one ear to the other and leaves the most uncomfortable procedure until last. In some cases, the ear canal may be too painful, swollen or full of exudate to allow a meaningful otoscopic examination to be performed. In these cases, the clinician has two choices. The animal can be sedated or anaesthetised, or a course of preliminary treatment can be given and the ear re-examined within a few days. Which course is taken will depend on the severity of the clinical presentation and clinician's index of suspicion for the various underlying causes. In either case, it is essential that a full otoscopic examination be performed at some stage.

In addition to performing an otoscopic examination, it is essential to perform cytological analysis of the exudate on the first and all subsequent visits. This can always be performed, even if the ear is too painful to allow a full otoscopic examination. Cytology allows preliminary differentiation of the types of infectious agents that may be present (cocci, rods or *Malassezia*). Detection of cocci or *Malassezia* allows empirical treatment whereas the presence of rods should prompt the clinician into performing bacterial culture and sensitivity testing. Cultures should also be performed on recurrent or resistant cases.

In addition to diagnosing and treating the otitis itself, the clinician should also commence efforts to determine the underlying cause. This is especially important in any case that has suffered otitis on more than one occasion. If this is not done, many cases will become chronic.

A summary of the diagnostic approaches to acute otitis (animals presenting for the first time) and chronic otitis (animals failing to respond to treatment or suffering multiple bouts of otitis) are shown in Figures 12.1 and 12.2 respectively. Further details on the treatment of otitis can be found in Chapter 14.

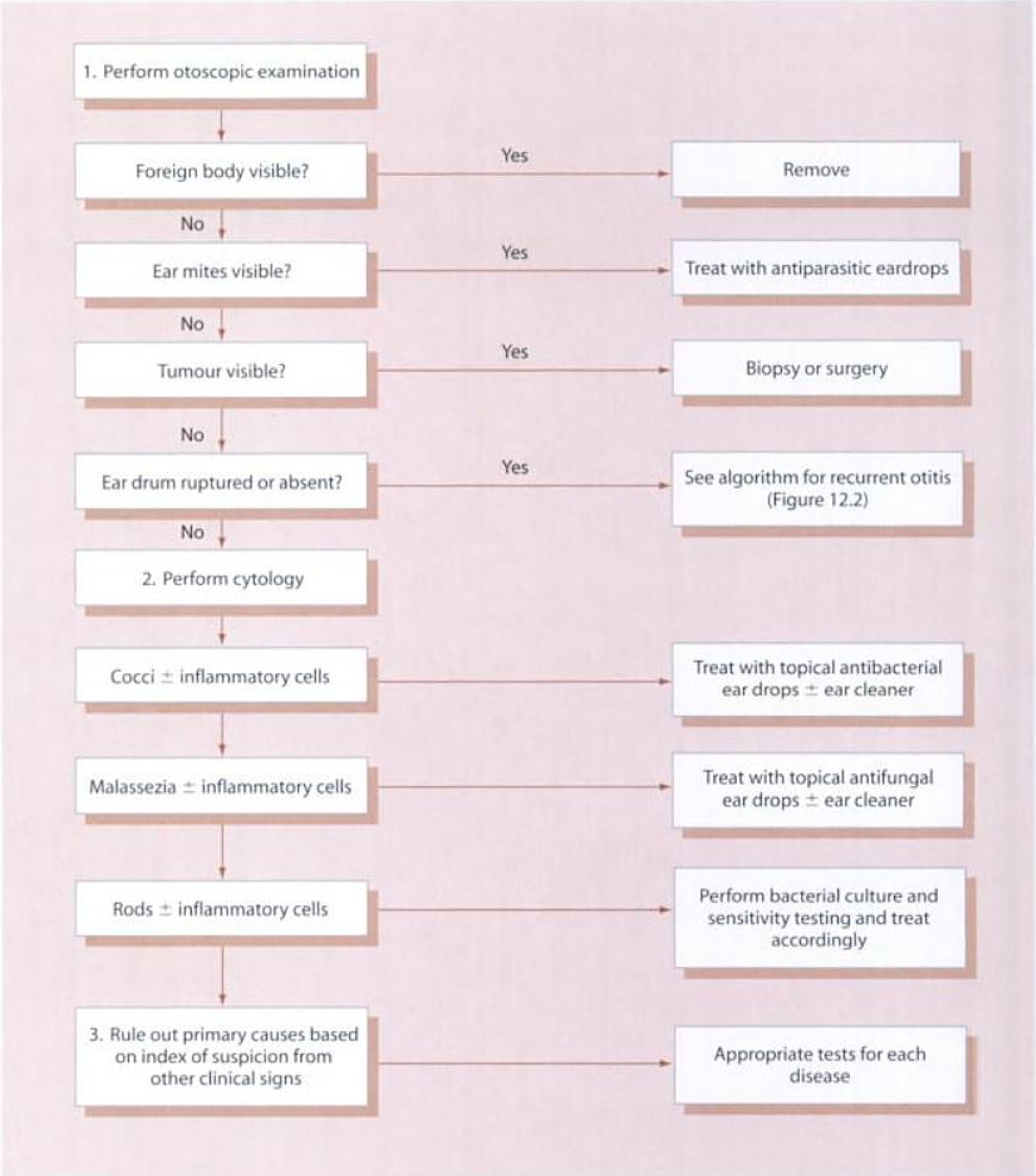

1. Perform otoscopic examination
Foreign body visible?
Yes
Remove
No
Ear mites visible?
Yes
Treat with antiparasitic eardrops
No
Tumour visible?
Yes
Biopsy or surgery
No
Ear drum ruptured or absent?
Yes
See algorithm for recurrent otitis (Figure 12.2)
No
2. Perform cytology
Cocci ± inflammatory cells
Treat with topical antibacterial ear drops ± ear cleaner
Malassezia ± inflammatory cells
Treat with topical antifungal ear drops ± ear cleaner
Rods ± inflammatory cells
Perform bacterial culture and sensitivity testing and treat accordingly
3. Rule out primary causes based on index of suspicion from other clinical signs
Appropriate tests for each disease

Figure 12.1 General diagnostic approach to acute otitis externa

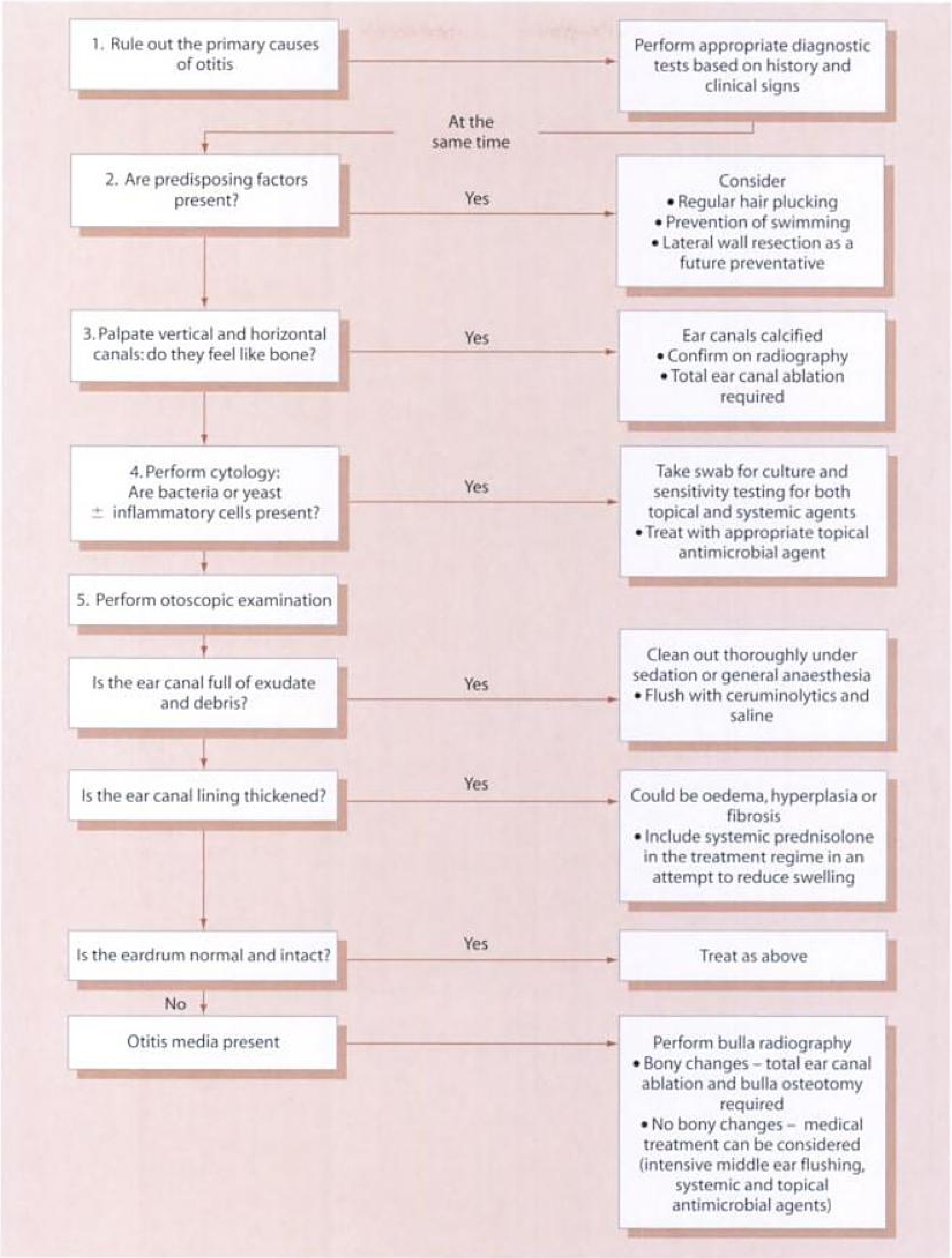

1. Rule out the primary causes of otitis
Perform appropriate diagnostic tests based on history and clinical signs
At the same time
2. Are predisposing factors present?
Yes
Consider
• Regular hair plucking
• Prevention of swimming
• Lateral wall resection as a future preventative
3. Palpate vertical and horizontal canals: do they feel like bone?
Yes
Ear canals calcified
• Confirm on radiography
• Total ear canal ablation required
4. Perform cytology: Are bacteria or yeast ± inflammatory cells present?
Yes
Take swab for culture and sensitivity testing for both topical and systemic agents
• Treat with appropriate topical antimicrobial agent
5. Perform otoscopic examination
Is the ear canal full of exudate and debris?
Yes
Clean out thoroughly under sedation or general anaesthesia
• Flush with ceruminolytics and saline
Is the ear canal lining thickened?
Yes
Could be oedema, hyperplasia or fibrosis
• Include systemic prednisolone in the treatment regime in an attempt to reduce swelling
Is the eardrum normal and intact?
Yes
Treat as above
No
Otitis media present
Perform bulla radiography
• Bony changes – total ear canal ablation and bulla osteotomy required
• No bony changes – medical treatment can be considered (intensive middle ear flushing, systemic and topical antimicrobial agents)

Figure 12.2 General diagnostic approach to chronic (recurrent or resistant) otitis. Clinicians must go through all the stages down the left-hand side in every case.

13

Performing and interpreting diagnostic tests

In the previous chapters, various diagnostic tests were listed for each of the conditions mentioned. This chapter describes how to perform and interpret those tests in order to yield diagnostically useful information. The information is divided into the following sections:

Equipment required for dermatological diagnosis 148
Using a microscope 149
Choosing and using an external laboratory 150
Specificity and sensitivity of diagnostic tests 152
Tests to find ectoparasites 152
Cytology 160
Trichography 176
Tests for dermatophytosis 179
Bacterial and fungal culture 182
Tests to investigate atopic dermatitis 185
Tests to investigate food allergy 190
Tests to investigate contact dermatitis 195
Biopsy and histopathology 198
Haematology (complete blood count) 204
Biochemistry (clinical chemistry) 206
Tests to assess thyroid function 212
Tests to assess adrenal function 221
Miscellaneous endocrine tests. 229

Equipment required for dermatological diagnosis

In order to perform dermatological diagnosis, the following equipment is required:

- A good quality binocular microscope – this is essential for detecting parasites, examining cytological specimens and performing trichography.
- Glass microscope slides – used for skin scrapings, cytology and trichography.
- Cover slips – essential for examining skin scrapings and cytology slides.
- Immersion oil – used for examining specimens under the 100× lens.
- Lens tissue and methylated spirit – used for cleaning microscope lenses.
- Magnifying glass – used for close examination of the skin and coat brushings.
- Flea comb – used for coat brushings.
- Variety of scalpel blades – used for skin scrapings and skin biopsy.
- Mineral oil – used for obtaining skin scrapings.

- Acetate tape – used for finding parasites and *Malassezia* organisms.
- Cotton tipped swabs (cotton buds) – used for collecting cytological specimens from the ears.
- Diff Quik stain – used for cytological examination.
- Fine tipped artery forceps – used for epilating hairs for trichography.
- Wood's lamp – used as a preliminary screening test for dermatophytosis.
- Toothbrushes – used for collecting material for dermatophyte culture.
- 20% KOH – used as a clearing agent when looking for dermatophytes.
- Dermatophyte test medium – used for culturing dermatophytes (only needed if dermatophyte cultures are performed in the practice laboratory).
- Lactophenol cotton blue – used for staining dermatophyte colonies (only needed if dermatophyte cultures are performed in the practice laboratory).
- Sterile culture swabs with and without transport medium – used for bacterial culture.
- Sterile containers – used for sending tissues to external laboratories.
- Syringes and needles – used for taking blood samples and performing fine needle aspirates.
- 4 mm, 6 mm and 8 mm biopsy punches – used for collecting skin biopsies.
- Basic surgical pack – used for collecting skin biopsies.
- Suture materials – used for closing skin biopsy sites.
- 10% neutral buffered formalin – used for fixing skin biopsy specimens.
- Cardboard – used for supporting skin biopsy specimens.
- Clippers – used for removing hair.
- Black felt-tip marker pen – used for marking biopsy sites and skin testing sites.
- Allergen set for intradermal testing (only appropriate for clinicians with a special interest in dermatology) – used for investigation of atopic dermatitis.
- Blood collection tubes – used for haematology, biochemistry and endocrine testing.

Using a microscope

The microscope is the single most important piece of equipment in the practice laboratory. Without a good quality microscope, accurate dermatological diagnosis is impossible. A binocular microscope is required with 4×, 10×, 40× and 100× lenses. With the 10× eyepieces, final magnifications of 40×, 100×, 400× and 1000× are obtained. It is important that the various settings of the microscope (field diaphragm, condenser focus knob, condenser centering screws) have been set up correctly. If the microscope has been recently purchased, it should have been optimally configured by the manufacturer on delivery. If not, it is well worth having it serviced because, with time, the settings on a microscope tend to change leading to sub-optimal performance.

The microscope should be placed in a fixed location in the laboratory on a bench or table at a suitable height in front of a comfortable stool or seat. Crouching over the microscope in a cramped corner of a room does not lend itself to slide viewing and can lead to neck or back discomfort. There should also be space around the microscope to store slides and immersion oil.

For optimal results, the clinician must prepare good specimens and examine them appropriately. With any sample, (e.g. skin scraping, cytological specimen, trichogram), it is important to use a cover slip. This provides a flat optical surface and greatly enhances the clarity of the image. For skin scrapings and trichograms, the cover slip is suspended on the mineral oil within which the sample is contained. For cytological specimens, a drop of immersion oil should be placed on the slide so that it covers the sample. The cover slip can then be suspended on top. For permanent mounting and storage of slides, the cover slip can be placed on a drop of DPX mountant (available from laboratory suppliers).

When viewing slides, it is important that the sample is neither too thick nor spread out too far over the slide. If too thick, the specimen is difficult to examine and diagnostic findings can be missed. If spread out too far, the slide takes longer to examine than it should, reducing working efficiency. With experience, it is possible to judge how much material should be put on a slide so that it can be properly examined under the area of a cover slip. Placing extra material outwith the area of a cover slip is non-productive because it can't be adequately examined.

When viewing skin scrapings and trichograms, it is useful to increase the contrast by turning down the condenser. This provides better definition of parasites and hairs against the pale background. When viewing cytological specimens (or histopathology slides) the condenser should be opened to allow as much light as possible to pass through the slide. This provides better clarity and detail of the stained cells. The light setting should be modified so that each individual slide is optimally illuminated.

When using the microscope, it is essential that the clinician has a good concept of scale. This allows the appropriate lens to be used for each sample. Figure 13.1 shows the size of various objects that are commonly seen during dermatological diagnosis and the lenses that should be used to find them.

To ensure optimum performance, it is important that the microscope is kept clean. After use each day, the lenses should be cleaned with lens tissue. The oil immersion lens should be cleaned with alcohol or methylated spirit and then dried. The microscope should then be covered with a dust cover.

Choosing and using an external laboratory

Some of the tests required for dermatological diagnosis need to be performed in specialist laboratories. These include culture and sensitivity testing of microorganisms, endocrine tests, immunological tests and histopathological diagnosis. Haematological and biochemical tests are also often performed by external laboratories. Most of the commercial laboratories offering these tests perform them to a high standard. However, some care is warranted in choosing a laboratory for dermatohistopathology. The interpretation of skin pathology has become increasingly specialised and it is advisable to send skin biopsies to a pathologist with a specific interest in dermatology. A large number of skin diseases can only be diagnosed histopathologically and many have been described relatively recently. Only dermatopathologists or pathologists with a specific interest in dermatology are likely to be familiar with all these conditions. There are a number of pathologists in the UK, USA, Canada, Europe and Australia who offer specialised dermatopathology services. The best way to find one is to consult a board-certified or specialist veterinary dermatologist.

Once the appropriate laboratories have been chosen, it is important to maximise the benefit that can be obtained from them. Clinicians must provide adequate documentation and samples in order to receive diagnostically accurate results. With objective tests (such as blood tests and hormone assays), the test result is independent of the animal's history and clinical signs. With these tests, only a limited amount of information is required by the laboratory. However, with other procedures (such as histopathological examination), the provision of an accurate description of the signalment, history and clinical signs is pivotal in making a definitive diagnosis. If clinicians do not provide this information, they cannot expect to receive consistently accurate results.

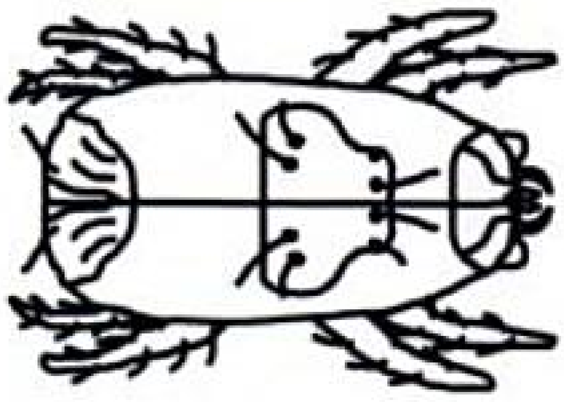
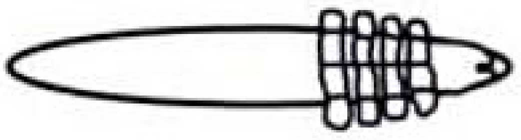
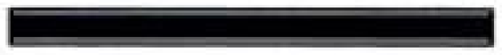
Objects (not to scale)	Actual size	Lens required	Apparent size after magnification
Round or oval mite 	300 μm in length	4×	1 cm
Demodex mite 	300 μm in diameter	4× or 10×	4× = 1 cm 10× = 2.5 cm
A hair 	30 μm in diameter	10× or 40×	10× = 0.3 cm in diameter 40× = 1.2 cm in diameter
Macrophage 	15 μm in diameter	100×	1.5 cm in diameter
Neutrophil 	10 μm in diameter	100×	1 cm in diameter
Malassezia 	4 μm in diameter	100×	0.4 cm in diameter
Coccus 	1 μm in diameter	100×	1 mm in diameter

Figure 13.1 Approximate size of objects seen during dermatological diagnosis

Clinicians should also ensure that the samples submitted are appropriately collected, stored, labelled and packaged. Sufficient material should be submitted to allow the required tests to be done. Laboratories are always willing to provide advice on sample submission and often provide free postage and packaging materials. Further information on collecting samples is provided later in this chapter.

Finally, clinicians should always correlate the test results from external laboratories with the clinical findings from the case. If the results do not fit with what is expected, the clinician should discuss the results with the laboratory. This is especially important with histopathology in which an incorrect diagnosis can lead to inappropriate treatment. Clinicians should never let the results of diagnostic tests erase a previous clinical suspicion. Accurate dermatological diagnosis relies on a critical level of clinicopathological correlation.

Specificity and sensitivity of diagnostic tests

Whenever a diagnostic test is performed, either within the practice or by an external laboratory, the clinician should be acutely aware of the concepts of specificity and sensitivity. These statistical parameters give an indication as to how reliable a test is in confirming or ruling out a particular diagnosis. Unless the clinician bears these concepts in mind, erroneous conclusions will be drawn from test results.

The specificity indicates the likelihood that a positive test result confirms a particular disease. The sensitivity indicates the likelihood that a test will be positive if the animal has the disease. To help understand these concepts, it is useful to look at two simple examples: the use of radiography to diagnose a broken leg and the use of rectal temperature to diagnose an infection. If an X-ray shows a fractured bone, it confirms that the leg is broken. Hence, the test is 100% specific because this finding can have no other explanation. Likewise, if an X-ray is taken of a broken leg, it will always detect the abnormality. The test is also therefore 100% sensitive. In an ideal world, all diagnostic tests would have 100% specificity and 100% sensitivity. Unfortunately, this is not the case. If an animal has an elevated rectal temperature, it may indicate that the animal has an infection. However, it could also indicate that the animal was excited, stressed, exercising or suffering from another non-infectious disease. Hence, the test is not specific 100% for infections. In addition, if an animal has an infection, its rectal temperature may or may not be raised. The test is therefore not 100% sensitive.

Many other tests, including those described in this chapter, lack specificity or sensitivity. The net result of this is that tests can give false positive or false negative results. If a test is not 100% specific, it will produce false positive results. Hence, even with a positive test result, clinicians should still consider alternative diagnoses. If a test is not 100% sensitive, the test will produce false negative results. The clinician should therefore still consider the suspected diagnosis and perform additional procedures to rule it out. This may include further diagnostic tests or trial therapies.

In this chapter, an approximate guide is provided as to a test's specificity and sensitivity for a particular disease. These figures are often estimates because the real figures are not known. As a guide to interpreting this information, the following summary will be useful:

Specificity – 100%:	The positive test result is diagnostic. The animal has the disease.
Specificity – <100%:	The positive test result is not always diagnostic. The animal may not have the disease.
Sensitivity – 100%:	If the animal has the disease, the test result will always be positive.
Sensitivity – <100%:	If the animal has the disease, the test result will not always be positive. The animal may still have the disease. The disease should be ruled out using other tests or treatments.

Tests to find ectoparasites

Coat brushing

A coat brushing is a simple diagnostic test in which a sample of scale and coat debris is examined both macroscopically and microscopically.

Technique	Simple
Interpretation	Simple
Specificity	100%
Sensitivity	≈ 50% (test may fail to detect the parasites)

Indications

Should be performed in any animal presenting with pruritus or scaling.
May be diagnostic in cases of:

- Flea infestations
- Pediculosis
- Cheyletiellosis

Technique

1. Brush the coat vigorously with a flea comb for 30 to 60 seconds. Try to obtain material from near the skin surface and avoid collecting too much hair.
2. Either brush the material directly onto a piece of paper or transfer the material from the comb onto the paper. White paper is preferable for detecting flea infestations and dark paper is preferable for detecting Cheyletiella and louse infestations.
3. Examine the collected material with the naked eye and/or with a magnifying glass.
4. Then, transfer the collected material onto a glass slide either by picking it up on a piece of adhesive tape (which is then stuck directly on the slide) or by tipping it into a drop of oil on the slide (which is then covered with a cover slip).
5. Examine under the low power (4×) objective of the microscope.

Examination

The initial material should be examined for signs of fleas or flea dirt. Fleas are laterally compressed, rapidly moving, wingless insects that jump considerable distances. They need to be killed in order to examine them microscopically. Flea dirt appears as dark specks of "soot-like" material. When transferred to a piece of moist cotton wool or gauze, the dark specks turn a reddish-brown colour, indicating the presence of dried blood.

The material should also be examined for the presence of lice which are visible to the naked eye or *Cheyletiella* mites which can be seen with a magnifying glass. After transferring the material to a glass slide, the specimen can be scanned on low power (4×) for flea dirt, lice, lice eggs, *Cheyletiella* mites or *Cheyletiella* eggs.

Unstained adhesive tape strip

A simple diagnostic test in which clear adhesive tape is used to collect superficial parasites from the superficial stratum corneum.

Technique	Simple
Interpretation	Simple
Specificity	100%
Sensitivity	≈ 50% (test may fail to detect the parasites)

Indications

Should be performed in any animal presenting with pruritus or scaling.
May be diagnostic in cases of:

- Pediculosis
- Cheyletiellosis
- *Trombicula* infestation

Technique

1. The area to be sampled should be free of hair. Gently clip if necessary but do not scrape the skin surface.
2. Repeatedly press a piece of clear adhesive tape onto the skin surface over an area of approximately 6 cm^2. Continue until the tape loses it's adhesiveness.
3. Stick the adhesive tape onto a glass microscope slide.
4. Examine under the low power (4×) objective of the microscope.

Skin scraping

A skin scraping is a simple diagnostic test in which a sample of the stratum corneum, epidermis and possibly the superficial dermis is examined microscopically for the presence of mites.

Technique	Simple
Interpretation	Simple
Specificity	100% (all mites)
Sensitivity	≈ 50% (surface mites), ≈ 99% (demodex mites)

Indications

Should be performed in any animal presenting with pruritus, alopecia, scaling, papulopustular eruptions, erosions, or draining tracts.

May be diagnostic in cases of:

- Demodicosis
- Cheyletiellosis
- *Sarcoptes scabiei* infestation
- *Notoedres cati* infestation
- *Otodectes cynotis* infestation
- *Trombicula autumnalis* infestation

Superficial skin scrapings

Superficial skin scrapings are useful for finding surface dwelling mites such as *Cheyletiella*, *Trombicula*, *Otodectes* and possibly *Sarcoptes* and *Notoedres*.

1. The area to be scraped should be free of hair. Clip if necessary. For superficial skin scrapings, a fairly large area can be sampled (up to 6 cm^2). Between two and six separate areas should be sampled to increase the diagnostic accuracy.
2. Mineral oil (liquid paraffin) is the preferred collection medium because it is possible to see whether the mites are alive or dead.
3. Place a small quantity of mineral oil on the skin surface.
4. Using a slightly blunted No. 10 or 20 scalpel blade, scrape the blade repeatedly across the skin surface over the entire area to be sampled (do not go deep enough to cause bleeding). At the culmination of each stroke, rotate the blade up and away from the skin surface so that the material is collected on the blade rather than just being pushed along the skin surface.

5. Transfer the sample from the blade onto a glass slide. This is easier if there is already a drop of mineral oil in the centre of the slide. Repeat steps 4 and 5 until an adequate sample has been obtained. Do not collect too much material or it will be too thick when examined under the microscope.
6. Mix the contents evenly so that the material is dispersed throughout the oil. However, do not spread the material over the entire slide because it will take longer to scan under the microscope. Place a cover slip over the collected material.
7. Examine the slide under the low power objective (4×). Use a bright light source but turn down the condenser to improve the contrast. All the listed mites can be seen under this magnification.

Deep skin scrapings

Deep skin scrapings are required for finding follicular parasites such as *Demodex* mites or *Pelodera* nematodes.

1. For deep skin scrapings, a smaller area should be sampled (up to 2 cm^2). The mites should easily be found in a single sample. However, multiple areas (up to five) should be sampled to determine if the problem is generalised.
2. Using a slightly blunted No. 10 scalpel blade, scrape the blade repeatedly across the skin surface over the area to be sampled. After the first few initial scrapes, squeeze the skin between thumb and forefinger to express mites from the follicles onto the surface. Then, continue scraping until capillary oozing is seen (the area should look like an abrasion). At the culmination of each stroke, rotate the blade up and away from the skin surface so that the material is collected on the blade rather than just being pushed along the skin surface.
3. Transfer the sample from the blade onto a glass slide. This is easier if there is already a drop of mineral oil in the centre of the slide. Repeat steps 4 and 5 until an adequate sample has been obtained. Do not collect too much material or it will be too thick when examined under the microscope.
4. Mix the contents evenly so that the material is dispersed throughout the oil. However, do not spread the material over the entire slide because it will take longer to scan under the microscope. Place a cover slip over the sample.
5. Examine the slide under the low power objective (4×). Use a bright light source but turn down the condenser to improve the contrast. Demodex mites can be seen quite easily under this magnification, but the 10× objective can be used for confirmation.

NB. *Some areas of the body are difficult to scrape (feet, eyelids). If these areas are affected, it may be possible to demonstrate demodex mites using a hair plucking technique (see Trichography). Also, in areas where there is extensive fibrosis (especially the interdigital regions) or excessive mucin deposition (especially SharPeis) it may be necessary to demonstrate demodex mites on histopathological examination of skin biopsies (see Skin biopsy and histopathology).*

Pustule examination for demodex mites

A method for determining if demodex mites are present in pustules or draining tracts.

Technique	Simple
Interpretation	Simple
Specificity	100%
Sensitivity	99%

Indications

Should be performed in any animal presenting with persistent or antibiotic-refractory pustules.

May be diagnostic in cases of:

- Demodicosis

Technique

1. Rupture the pustule with a needle.
2. Press a glass slide against the pustule contents. Let the sample dry, but do not stain.
3. Place a drop of mineral oil on the slide and float a cover slip on top.
4. Examine under the low power (4× or 10×) objectives of the microscope. Use a bright light source but turn down the condenser to improve the contrast.

Ear smear for otic parasites

A method for determining if parasites are present in the ear canal.

Technique	Simple
Interpretation	Simple
Specificity	100%
Sensitivity	≈ 80% (test may fail to detect the parasites)

Indications

Should be performed in any animal presenting with otitis.

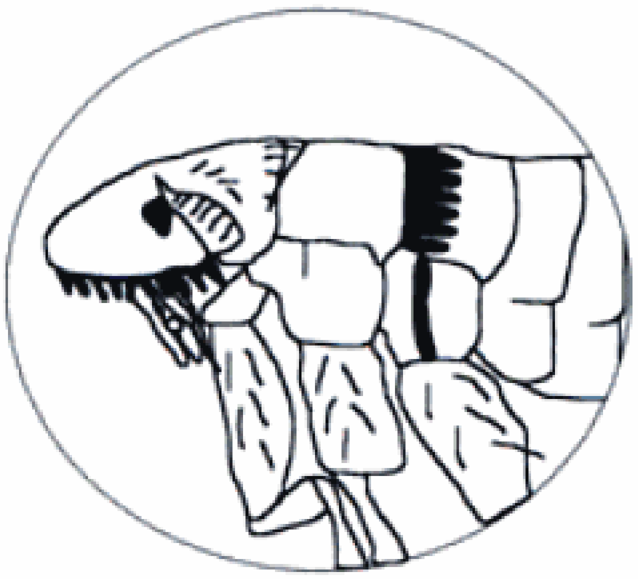
May be diagnostic in cases of:

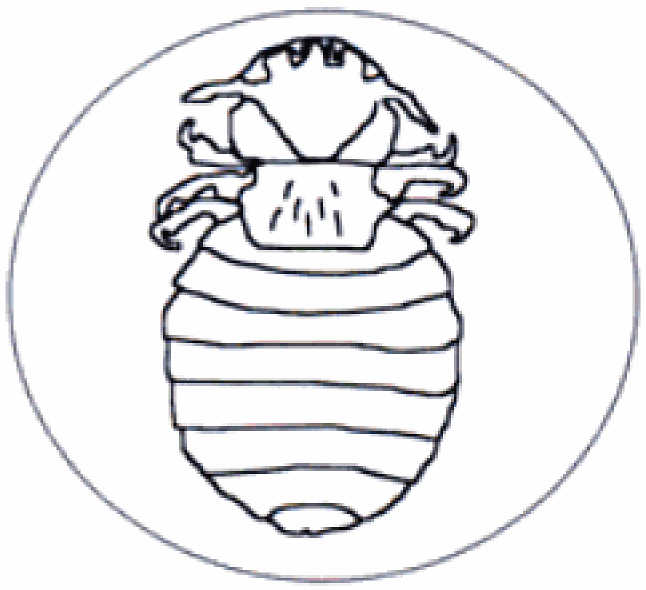
- *Otodectes cynotis* infestation
- Otic demodicosis

Technique

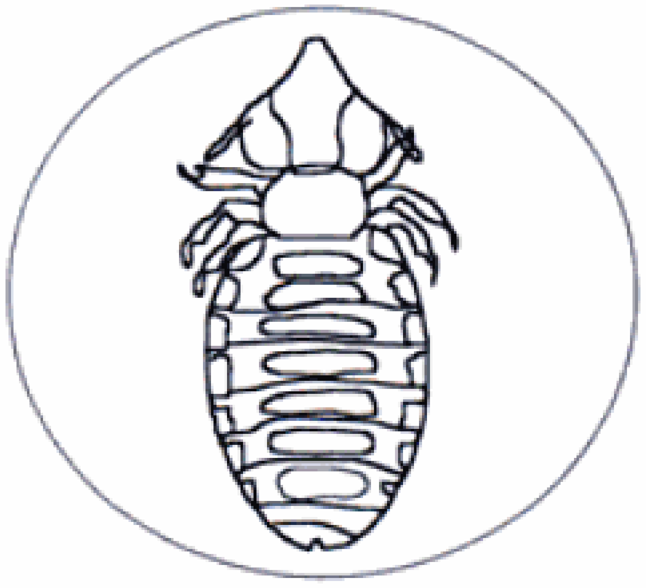
1. Collect a sample of the aural exudate with a cotton tipped swab.
2. Transfer the material to a glass slide containing a drop of mineral oil. Mix the contents to remove as much material from the swab as possible.
3. Place a cover slip on top.
4. Examine under the low power (4×) objective of the microscope. Use a bright light source but turn down the condenser to improve the contrast.

Interpreting parasitological tests

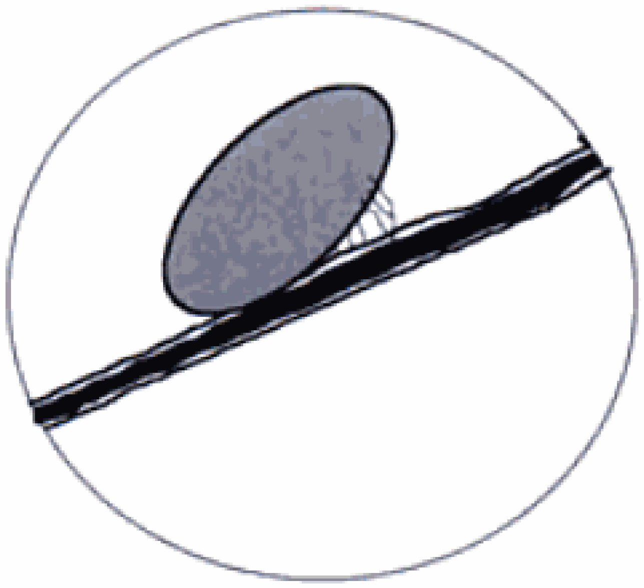
Appearance	Interpretation
	This field contains the front end of a flea (*Ctenocephalides felis*). Various species of fleas may be found on dogs and cats. They are distinguished by the appearance of the combs situated around the head. For details on further identification, consult parasitology textbooks. (Low power)



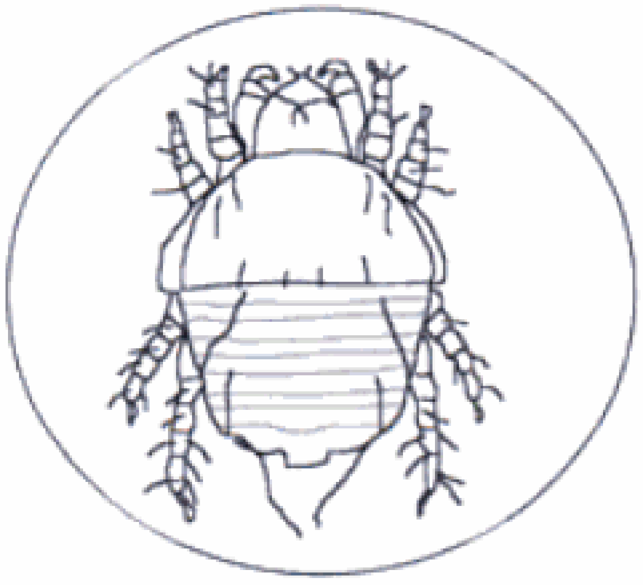
This field contains a dog louse. Lice have six legs, are flattened dorso-ventrally, have no wings and are host-specific. Three species of lice can infest dogs. The commonest is *Trichodectes canis* (shown here).The other two are *Linognathus setosus* and *Heterodoxus spiniger*, but these are uncommon. For details on identification, consult parasitology textbooks.
(Low power)



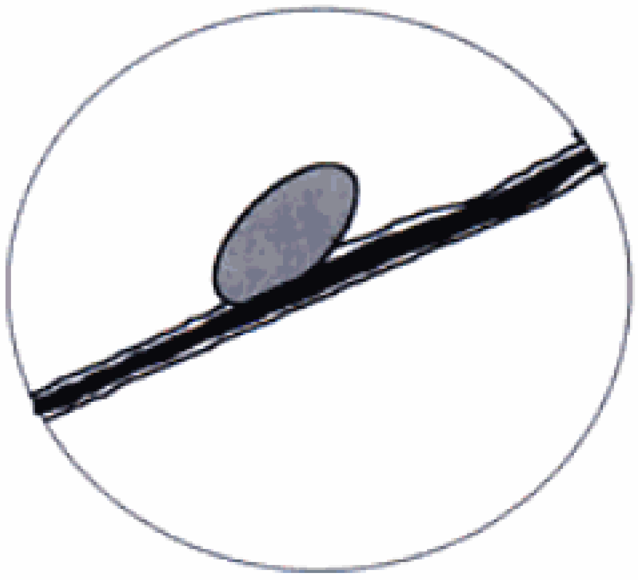
This field contains a cat louse (*Felicola subrostrata*). This is the only louse species to infest cats.
(Low power)



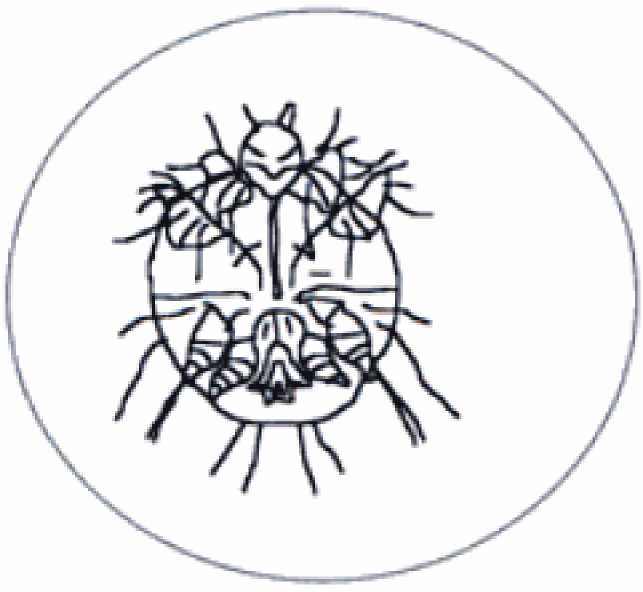
The field contains a hair with an attached louse egg. Lice eggs are visible to the naked eye (nits) in contrast to *Cheyletiella* eggs which are microscopic.
(Low power)



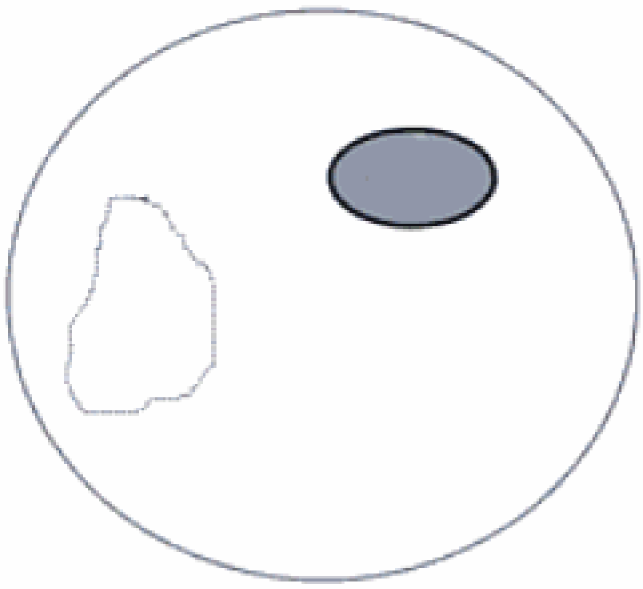
This field contains a large mite. The mite is oval with a broad shoulder region. The eight legs extend well beyond the body and there are easily visible hooks at the cranial end (accessory mouthparts). This is the typical appearance of *Cheyletiella* mites which may be found on dogs, cats and rabbits. The presence of a single mite confirms the diagnosis of cheyletiellosis.
(×100 magnification)



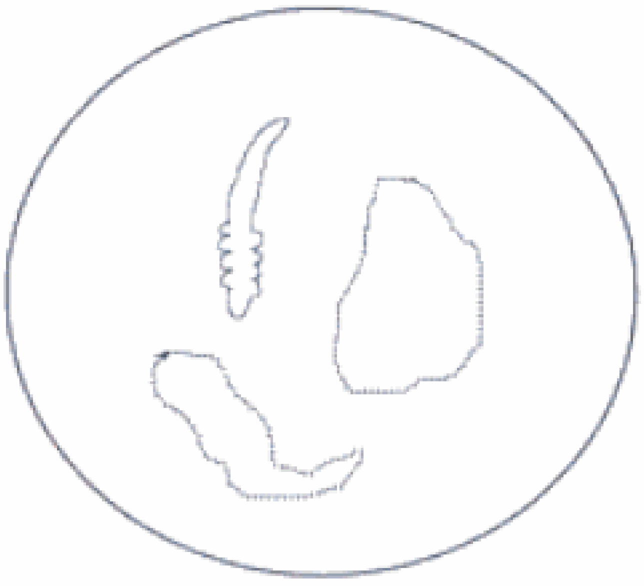
This field contains a hair with an attached *Cheyletiella* egg. *Cheyletiella* eggs are smaller than lice eggs.
(×100 magnification)



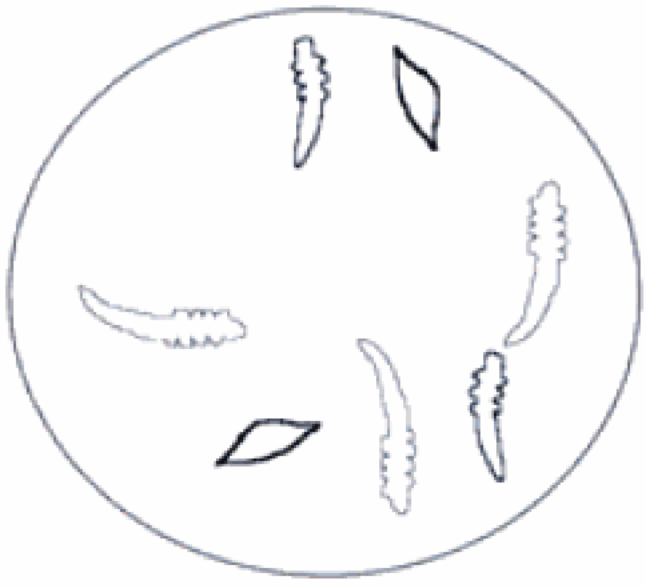
This field contains a round mite that has eight short stumpy legs which do not extend far beyond the edge of the body. Spines may be visible over the dorsal aspect. This appearance is typical of *Sarcoptes scabiei* (dog) or *Notoedres cati* (cat). The presence of a single mite confirms the diagnosis of scabies. (×100 magnification)



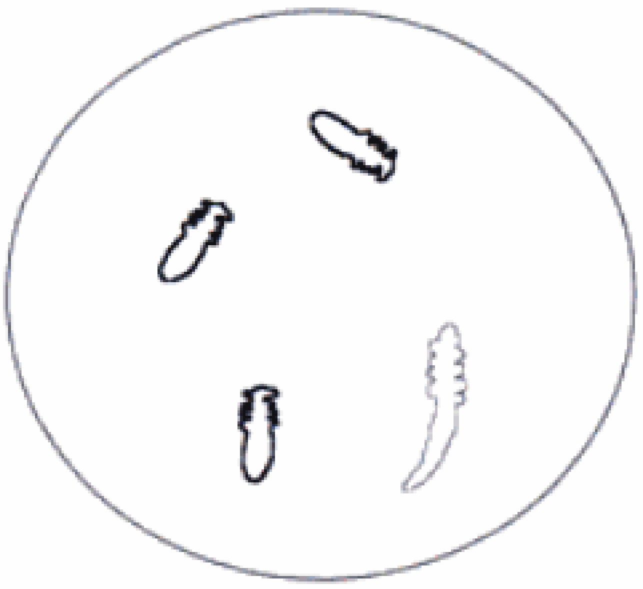
The field contains a surface corneocyte and an egg. An oval shaped egg found on the skin surface is most likely to be from *Sarcoptes scabiei*. *Cheyletiella* and lice eggs are usually attached to hairs, flea eggs fall off the host and demodex eggs have an elongated shape (see below). The presence of a single egg confirms the diagnosis. (×100 magnification)



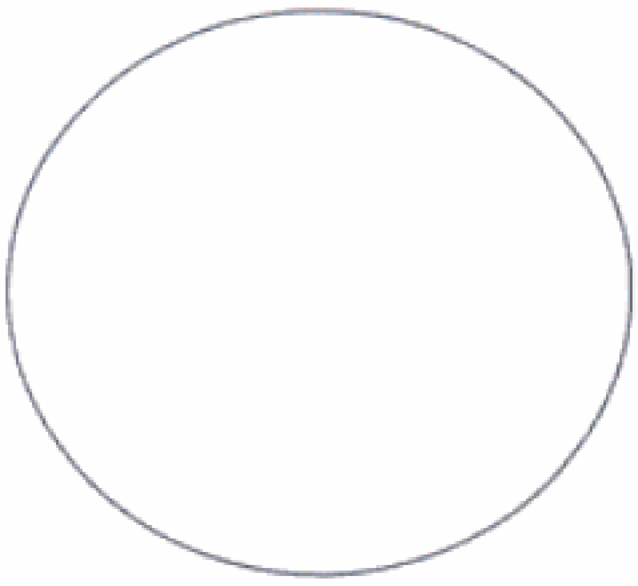
This field contains some surface corneocytes and a single demodex mite. The mite is long, thin and has eight stumpy legs. The length of the tail region is as long as the thoracic region. This appearance is typical of *Demodex canis* (dog) or *Demodex cati* (cat). Although demodex mites are a normal inhabitant of skin, they are virtually never found on skin scrapings from normal animals. This appearance warrants further scanning of the slide. If no more mites are found, further deep skin scrapings should be taken and examined. (×100 magnification)



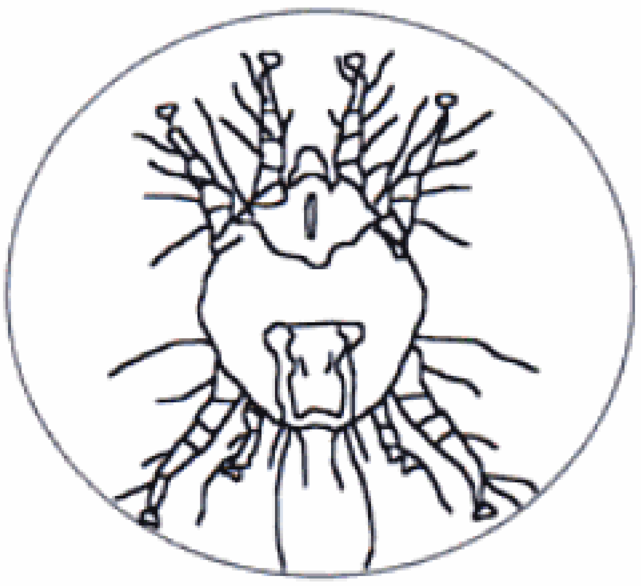
The field contains three eight legged demodex mites, two six legged nymphs and two "lemon-shaped" eggs. Seeing numerous examples of any of the life cycle stages is diagnostic for demodicosis. (×100 magnification)



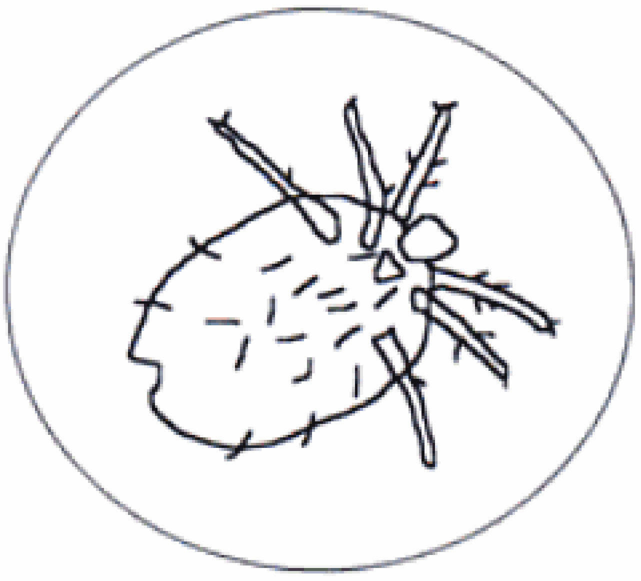
The field contains one long and three short demodex mites. The short mites have eight stumpy legs but the tail region is much shorter than the thoracic region. This appearance is typical of the short form demodex mite seen in dogs (un-named) and *Demodex gatoi* (cats). These mites live in the superficial layers of the stratum corneum rather than in the hair follicles and can therefore be found on superficial skin scrapings. These mites are often found in conjunction with excessive numbers of the long form.
(×100 magnification)



This field contains a single, degenerate, "ghost-like" demodex mite. This is often seen during treatment as the mites are being killed. If dead mites are found, the treatment should continue until they disappear completely.
(×100 magnification)



This field contains a large oval mite with eight long legs. Numerous sensory hairs project from the legs, which possess suckers at the end of stalks. This is the typical appearance of *Otodectes cynotis*. This mite is usually found in the ears but on rare occasions it can be found on the skin surface.
(×100 magnification)



The field contains a mite-like organism with six legs. This is the larval stage of *Trombicula autumnalis* and is only seen in certain geographic locations at certain times of year.
(×100 magnification)

ELISA to detect anti-scabies antibodies

An enzyme-linked immunosorbent assay (ELISA) has been developed to detect IgG antibodies directed against *Sarcoptes scabiei*. This allows scabies to be diagnosed with a blood test.

- Cotton swabs
- Fine needle aspiration

Which method is used depends on the type of lesion to be sampled. Dry, greasy, or scaly lesions are best sampled using clear adhesive tape. Moist lesions, pustules, purulent exudates, or ulcers are best sampled using impression smears. Cotton swabs can be used to obtain samples from difficult to reach areas such as the ear canals. Fine needle aspiration is used to obtain samples from solid lesions such as lumps and swellings.

Clear adhesive tape

1. The type of tape used is very important because there is quite marked variation in both the adhesive properties and the degree of transparency/graininess between brands. "Cellux" and "Diamond Clear" tape are both useful. If these are not available in a particular area, it is worth experimenting with several different types of tape to find one that is satisfactory.
2. Cut a piece of tape slightly longer than the length of a glass slide.
3. Press the middle of the tape repeatedly onto the lesional skin surface until it loses its adhesiveness. This means that it is covered in corneocytes.
4. Stick one end of the tape onto a glass slide to facilitate staining.

Impression smears

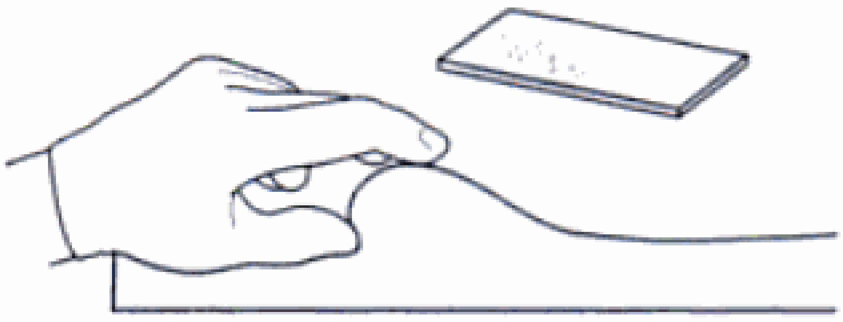
1. Press a glass slide onto the surface of the lesion or exudate. Do not repeatedly press the slide onto the surface because the sample obtained may become too thick. If you wish to collect progressively deeper samples from the same lesion, move the slide along each time or use a new slide for each sample.
2. To sample an intact pustule, rupture the pustule with a needle and then press the slide onto the surface as above.
3. Air-dry the slide before staining.

Cotton swabs

1. Place the cotton swab into the ear canal and gently remove some of the exudate or wax.
2. Roll the swab over the surface of the slide. Do not rub the swab backwards and forwards because this will disrupt the cellular components.

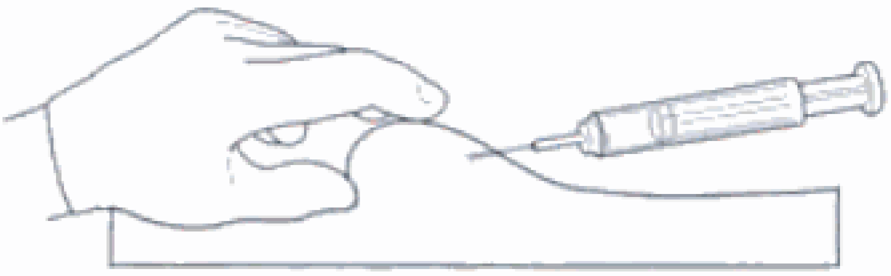
Fine needle aspirates

1. Spray or wipe the surface of the mass with alcohol. Hold the mass to fix its position

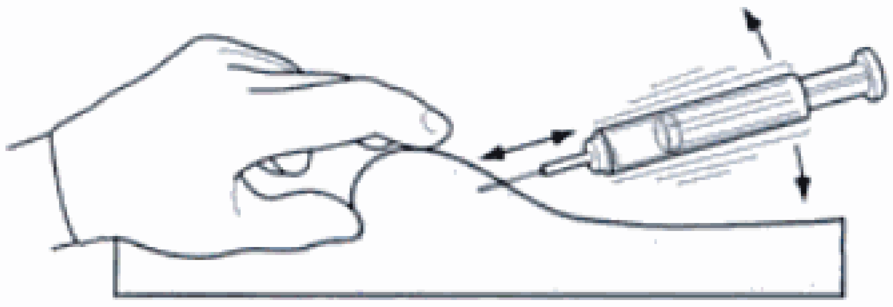


2. Insert a 20-gauge needle attached to a 5 ml syringe.

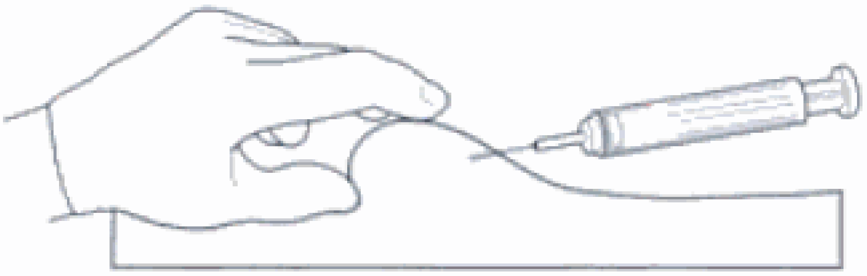
3. Withdraw the plunger to produce negative pressure.



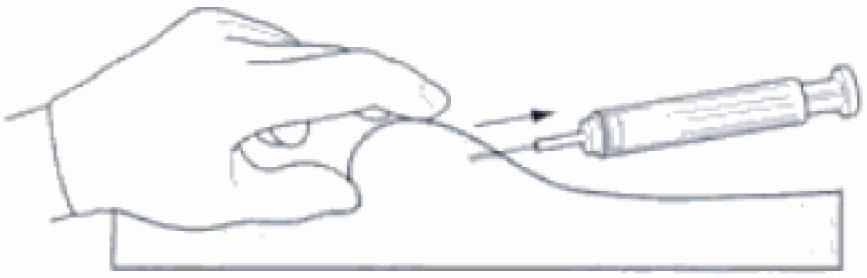
4. Move the needle around a few times, backwards and forwards, up and down and side to side. Do not let the needle leave the mass.



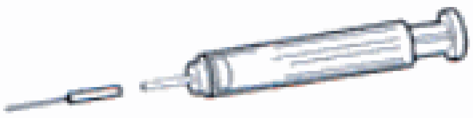
5. Release the suction on the plunger



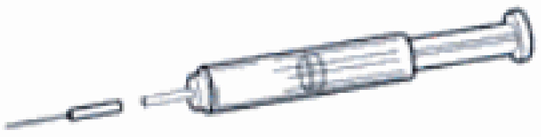
6. Withdraw the syringe and needle from the mass. Then, perform steps 7 to 10 very rapidly (less than 3 seconds) so that the material does not dry out within the needle



7. Remove the needle from the syringe.

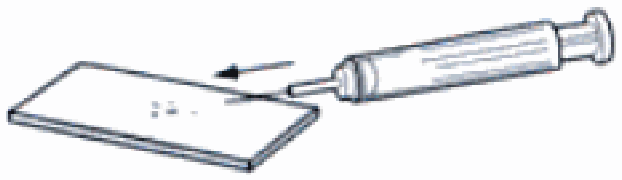


8. Fill the syringe with air.



9. Reconnect the needle

10. Forcibly expel the contents onto a glass slide. Check the slide carefully to see if there is any material on it (there may be a very small amount). Air-dry the slide in preparation for staining. If the technique does not work first time, it should be tried again. If it still doesn't work, the technique can be tried with a needle without the syringe attached. The syringe is then just used to blow the contents out onto the slide.



Staining and examining cytology samples

For most routine clinical purposes, the most useful staining system is Diff Quik (Dade). This comprises a methanol fixative, a red eosin dye and a blue thiazine dye.

1. For general staining, dip the slide or adhesive tape into the fixative five times. Then dip the slide or tape into the red dye five times followed by the blue dye five times. At each stage, each dip should last 1 second.
2. Rinse the slide or tape thoroughly with distilled water.
3. If using tape, fold it back over the glass slide (sticky side down). As the sample is protected underneath the tape, the slide may be dried by pressing it between tissue paper. If using glass slides, the sample should be air-dried. Do not dry with tissue paper or the sample may be removed.
4. When dry, adhesive tape strips can be examined immediately. For glass slides, the sample should be covered with a cover slip in order to improve the clarity when examining under the microscope. Place a drop of immersion oil over the sample and float the cover slip on top. Adhesive tape acts as its own "cover slip" so an additional cover slip is not required.
5. Scan the whole slide on low power and look for areas that have stained purple. Then look at these areas under high power (×400) or oil immersion (×1000).

NB. *It should be noted that there are two situations when this standard staining routine should be modified:*

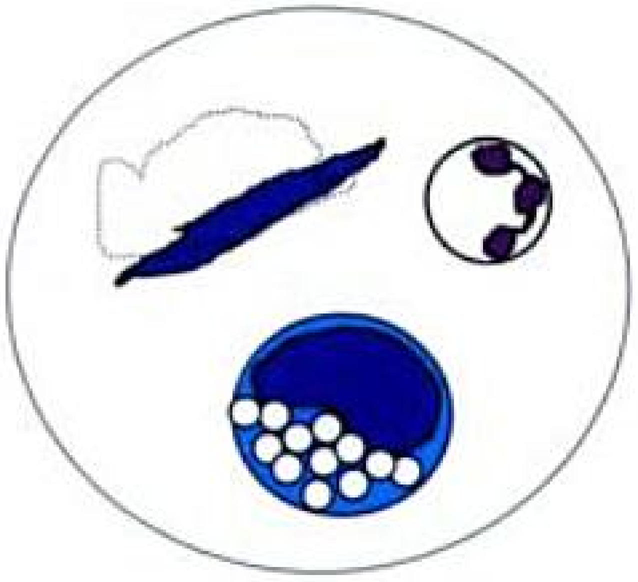
- *If the sample is predominantly oily or waxy (lipomas, ear wax, very greasy skin lesions), the methanol fixative may dissolve the sample. In these situations the fixative can be avoided altogether (if using tape) or the slide can be heat-fixed (impression smears, cotton swabs or fine needle aspirates). To heat fix the slide, put the slide through a Bunsen or lighter flame four or five times until it is too hot to touch. After cooling, stain the slide using only the red and blue dyes.*
- *Occasionally, the blue dye may fail to stain the slide adequately. This usually means that the dye needs changing. However, the intensity of blue staining can be increased by placing the slide in the blue dye for longer periods.*

Interpreting cutaneous cytology

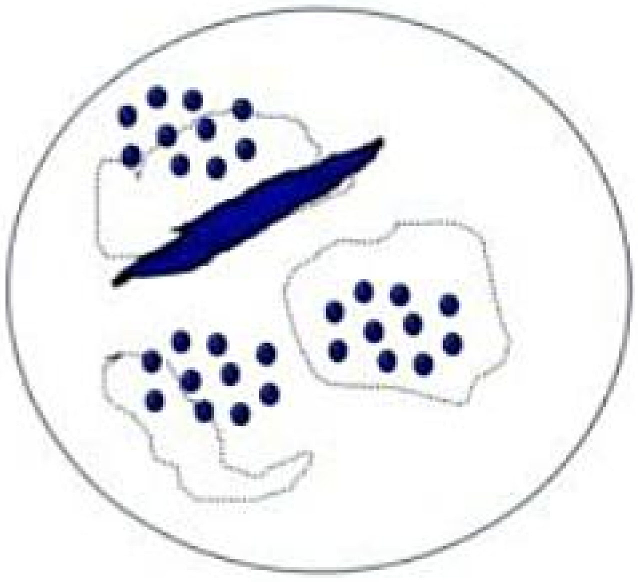
As with the interpretation of any diagnostic test, it is important to know what can be expected normally before deciding if something is pathological.

Normal cytological findings	Pathological findings
Surface corneocytes, hairs, occasional bacteria and Malassezia organisms, melanin, granules, fungal contaminants, stain precipitate.	Abnormal or excessive numbers of microorganisms
	Inflammatory cells (neutrophils, macrophages, eosinophils, lymphocytes, plasma cells, mast cells).
	Epidermal cells from below the stratum corneum (acantholytic keratinocytes)
	Neoplastic cells or cyst contents

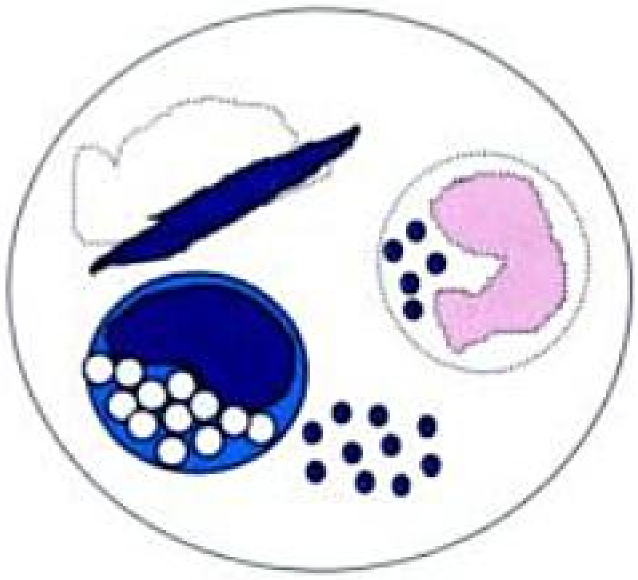
When interpreting cytological preparations, the observer must first distinguish between those findings that are found normally and those that represent disease. Then, it should be determined whether the pathological changes represent inflammation or neoplasia. In



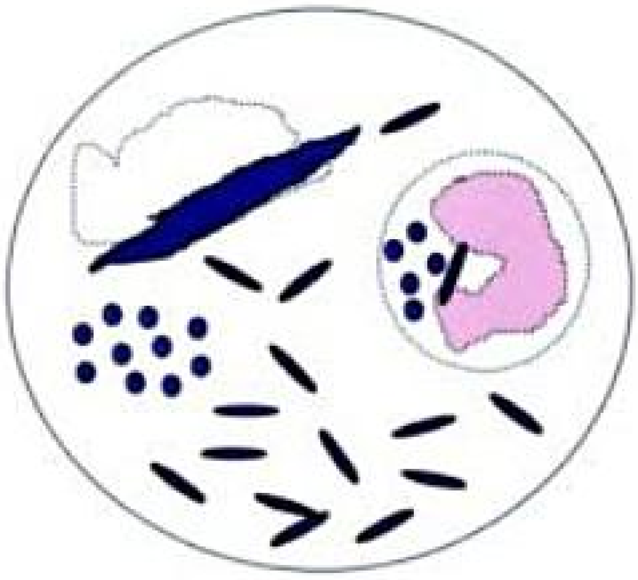
In this field, there is a neutrophil and a macrophage. However, the cytoplasm of the macrophage is filled with non-staining vacuoles. This indicates that the macrophage is activated. This is a common finding in chronic infections.



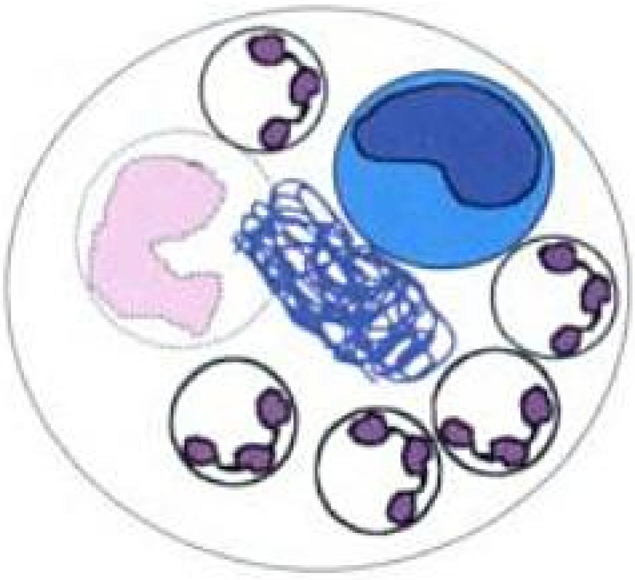
In this field, there are large numbers of blue staining extracellular cocci. This may indicate staphylococcal infection but could also represent surface contamination. The slide should be scanned for further evidence of active infection before pyoderma is diagnosed. Note that Diff Quik staining does not allow the organisms to be referred to as Gram positive or Gram negative.



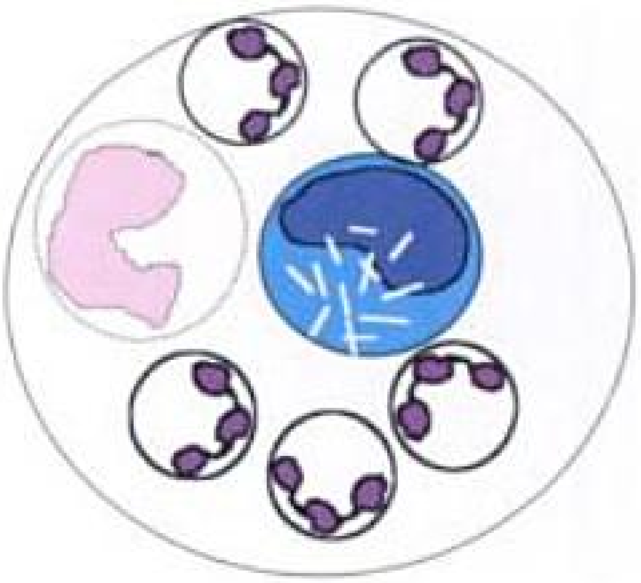
This field contains extracellular cocci, a degenerate neutrophil and an activated macrophage. The neutrophil contains phagocytosed cocci. This picture is diagnostic for staphylococcal pyoderma. However, intracellular cocci are not found as frequently as might be expected so the slide needs to be scanned carefully to find them.



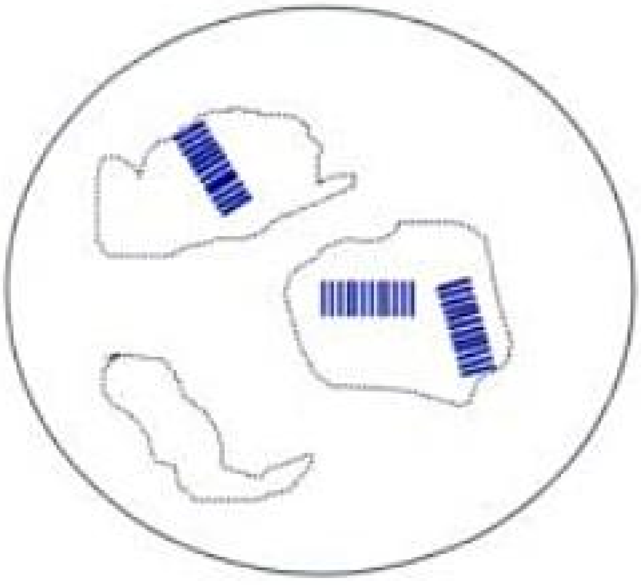
This field contains a mixed population of cocci and numerous rods. Some of the bacteria are present within a degenerate neutrophil. This would be a rare finding on skin cytology but is commonly seen in ear infections and cat bite abscesses. When this picture is seen in the ear, culture and sensitivity testing is indicated.



In this field a focus of pyogranulomatous inflammation containing neutrophils and a macrophage is surrounding a mass of mycelial hyphae. The hyphae usually stain blue. This appearance is suggestive of nocardiosis, actinomycosis or eumycotic mycetoma. Culture of the draining exudate would be indicated.

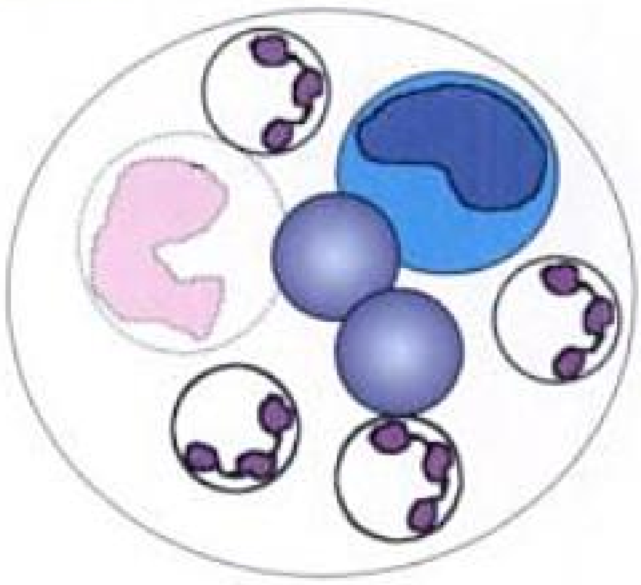
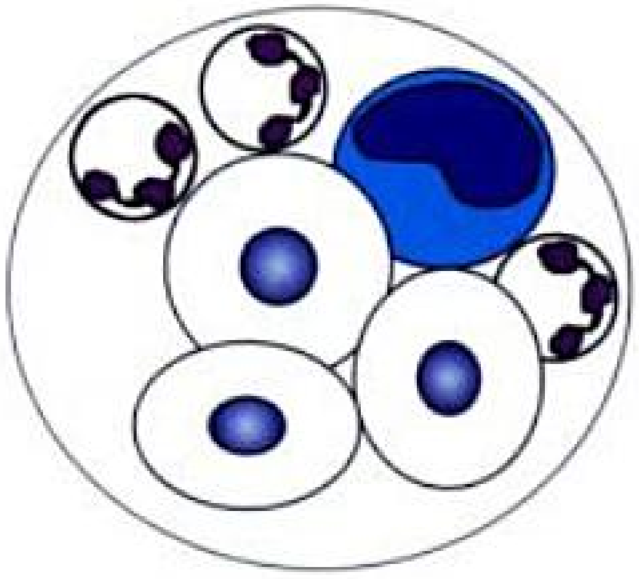


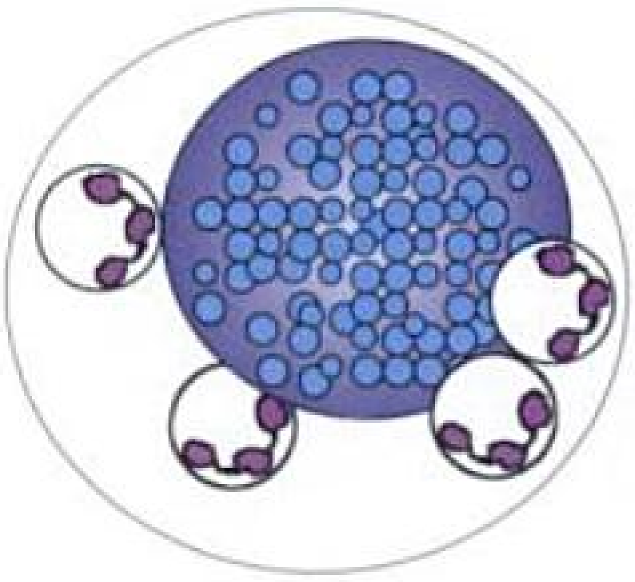
In this field, a focus of pyogranulomatous inflammation surrounds a macrophage containing clear rods. This rare finding would be suggestive of mycobacterial infection and the sample should be stained with an acid fast stain such as Ziehl-Neelson.



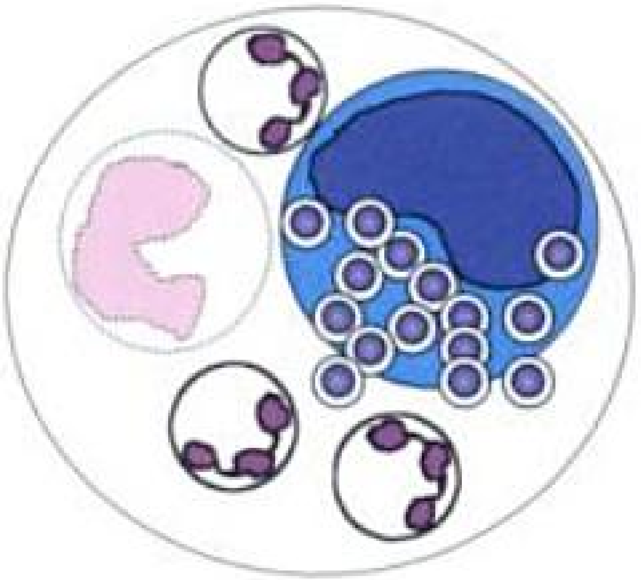
This field shows some *Simonsiella* organisms. They are large, darkly staining rods that are positioned side by side to give the appearance of a much larger, banded organism. *Simonsiella* are normal inhabitants of the oral cavity and they are transferred to the skin by licking. Their presence does not indicate skin infection but suggests pruritus.

Deep fungal infections (these are rare and have specific geographical distributions)

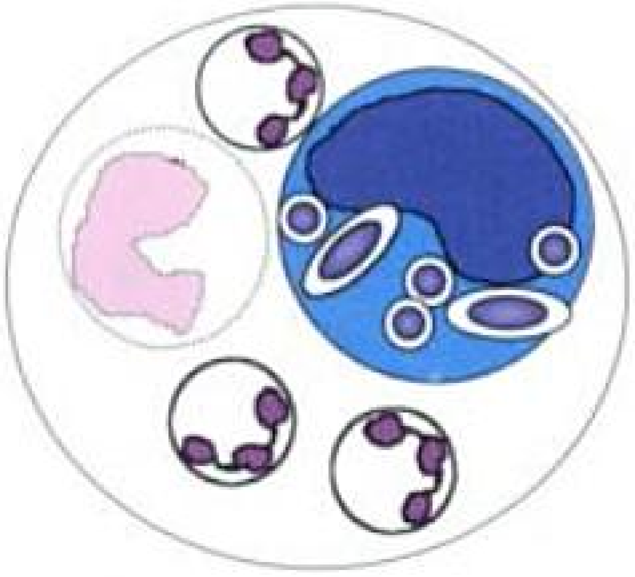
Appearance (×1000)	Interpretation
	This field contains a broad based, budding, thick walled, deeply basophilic yeast surrounded by pyogranulomatous inflammation. This is the characteristic appearance of *Blastomyces dermatitidis* and the finding would confirm a diagnosis of blastomycosis.
	This field contains three, round, basophilic yeasts with a large, non-staining capsule. This is the characteristic appearance of *Cryptococcus neoformans* and the finding would confirm a diagnosis of cryptococcosis.



This field contains a large, deeply basophilic spherule that contains large numbers of endospores. This is the characteristic appearance of *Coccidioides immitis* and the finding would confirm a diagnosis of coccidiomycosis.

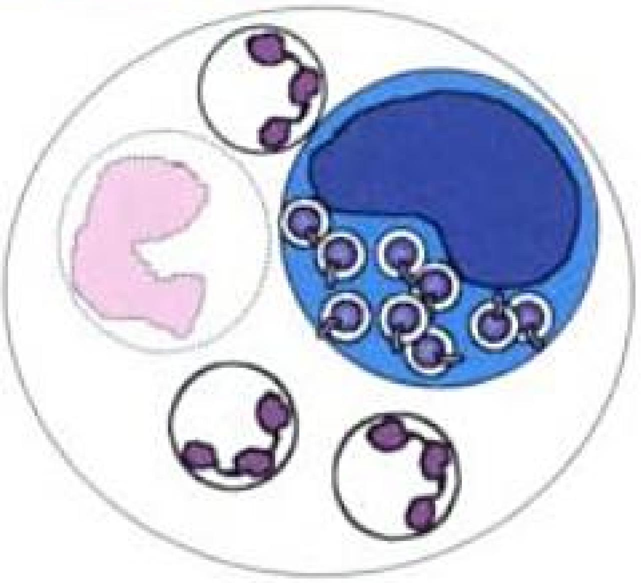


The macrophage in this field contains large numbers of small, round bodies with a basophilic centre and pale halo. This appearance would be consistent with *Histoplasma capsulatum.*



The macrophage in this field contains a number of round to cigar shaped bodies with a basophilic centre and a pale halo. The appearance of these organisms is consistent with *Sporothrix schenckii.*

Protozoal infections

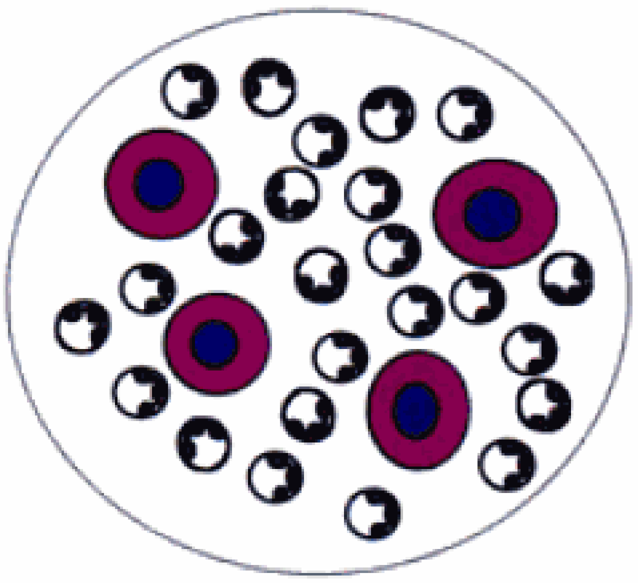


The macrophage in this field contains a number of round organisms with a basophilic centre and a small protruding rod (the kinetoplast). The appearance of these organisms is consistent with Leishman-Donovan bodies caused by *Leishmania* spp.

***NB** The appearance of* Histoplasma, Sporothrix *and* Leishmania *organisms is quite similar and care should be taken in distinguishing between them, especially if all three infections are known to occur in a particular area.*

Neutrophilic inflammation with acantholysis

Appearance (×400)



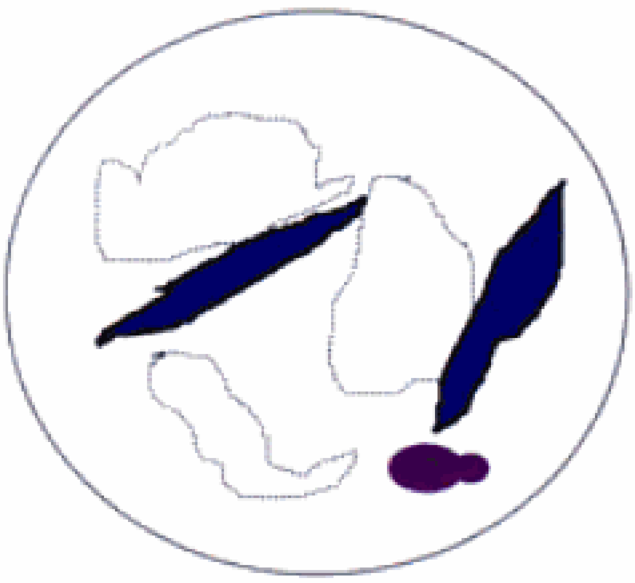
Interpretation

Acantholysis refers to a process in which the keratinocytes from deeper layers within the epidermis become separated from each other. The separated cells are known as acantholytic keratinocytes or acanthocytes. Acanthocytes are very large cells that are about five times the diameter of a neutrophil with a centrally placed nucleus. The presence of large numbers of acanthocytes amongst a background of neutrophils is a characteristic finding in pemphigus foliaceus. However, culture of pustules and biopsy is indicated whenever this picture is seen because bacterial infections can occasionally cause this pattern of inflammation.

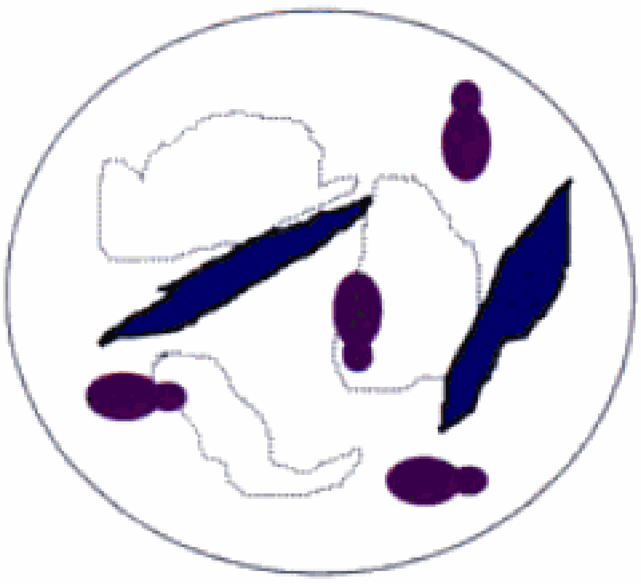
Malassezia dermatitis and otitis

Appearance (×1000)

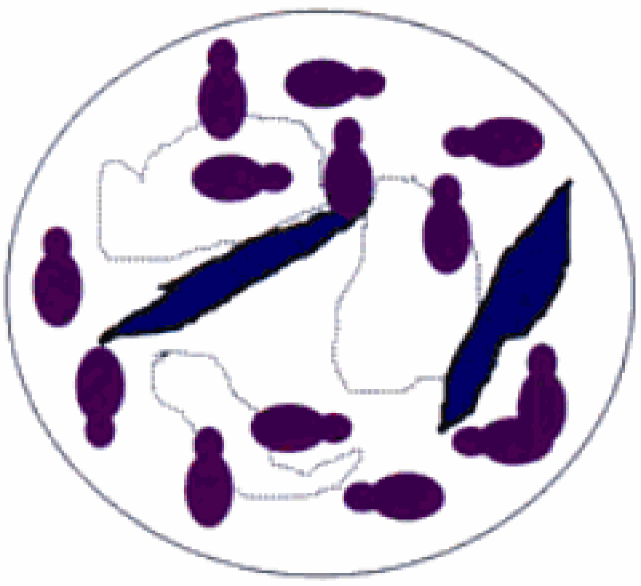
Interpretation



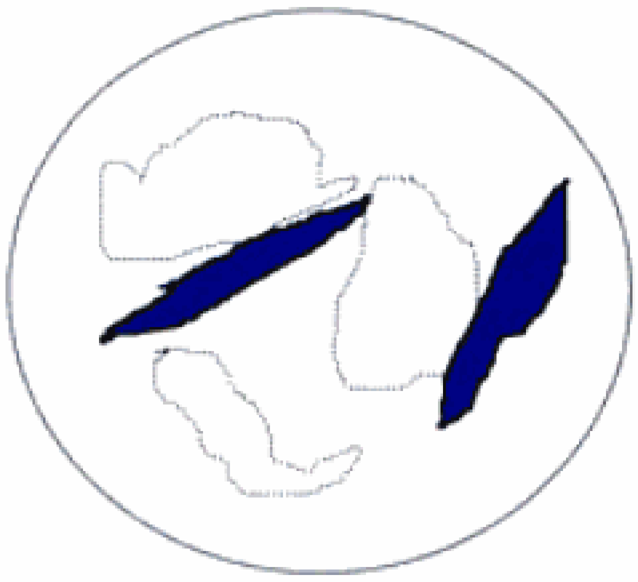
This field contains a single Malassezia organism in addition to some corneocytes. This could be consistent with normal skin. However, if the majority of the fields had this appearance, then it could be consistent with Malassezia overgrowth. If the clinical signs were consistent, trial therapy would be indicated.



The field contains four Malassezia organisms in addition to the corneocytes. If there are many fields with this appearance, there is a Malassezia overgrowth. Treatment is indicated. Note that Malassezia overgrowth does not usually lead to the presence of inflammatory cells on the skin surface.

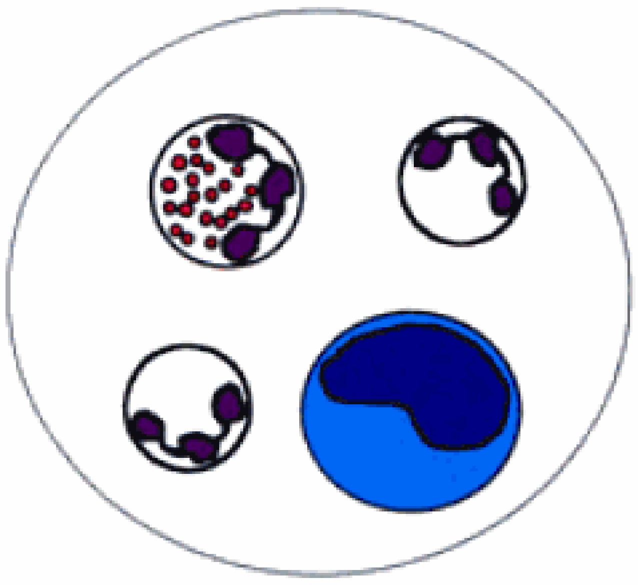
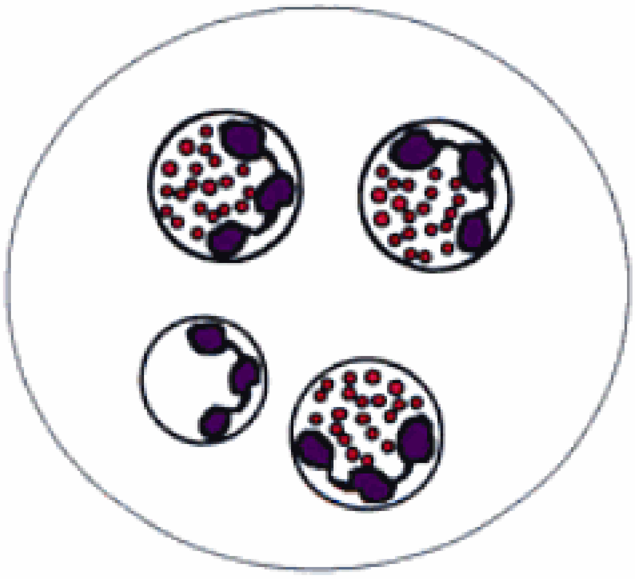
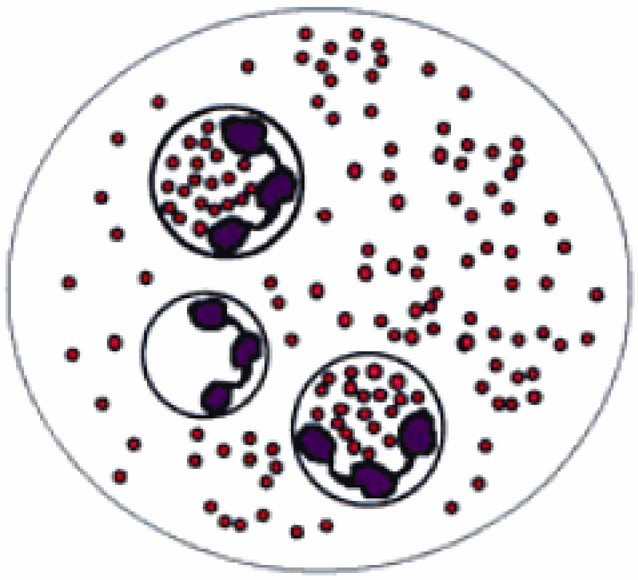


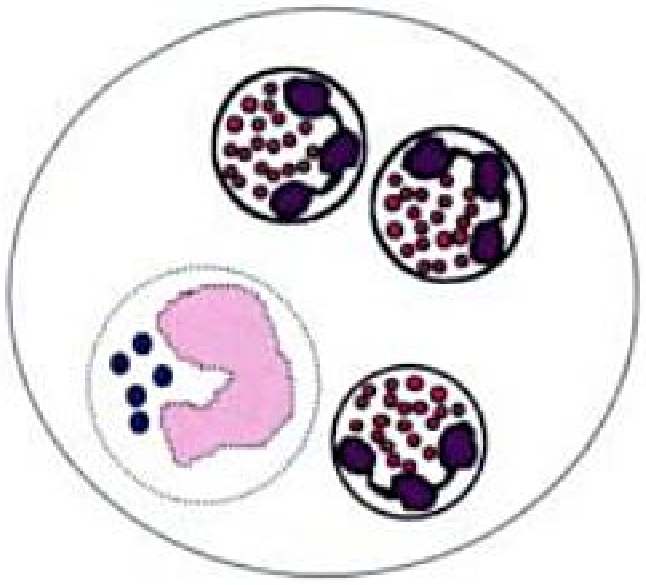
Highly significant numbers of Malassezia organisms. This is occasionally seen on the skin but more commonly in the ear. Treatment is indicated.



Four Malassezia organisms are present but they are very pale staining ("ghosts"). This occurs when the organisms fail to take up the blue stain. Slides need to be evaluated very carefully to avoid missing these organisms. A further sample should be obtained and placed in the blue stain for a longer period.

Eosinophilic inflammation

Appearance (×1000)	Interpretation
	This field contains two neutrophils, a macrophage and an eosinophil. The eosinophil is similar in appearance to the neutrophil but its cytoplasm is filled with pinkish-red granules. These can be confused with bacteria, but cocci stain blue with Diff Quik rather than red. Occasional eosinophils can be found in any inflammatory skin disease.
	This field contains three eosinophils and a neutrophil. When eosinophils make up a significant proportion of the cellular infiltrate (approaching 50% or more), the inflammation can be described as eosinophilic. This usually indicates either a parasitic infestation, or in cats, lesions of the eosinophilic granuloma complex (especially the eosinophilic plaque).
	This field contains two eosinophils and a neutrophil. In addition, the background contains a mass of pinkish-red granules. This indicates that some of the eosinophils have degranulated, releasing inflammatory mediators into the extracellular milieu. These granules are often confused with bacteria but they are the wrong colour for Diff Quik stain.

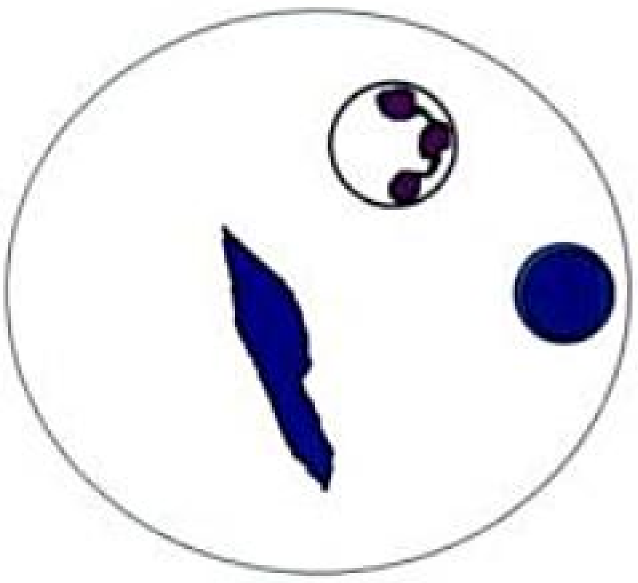


This field contains three eosinophils with some intercellular eosinophilic granules. In addition, there is a degenerate neutrophil containing intracellular cocci. This confirms the concurrent presence of eosinophilic inflammation and secondary bacterial infection. This is often seen in eosinophilic plaques.

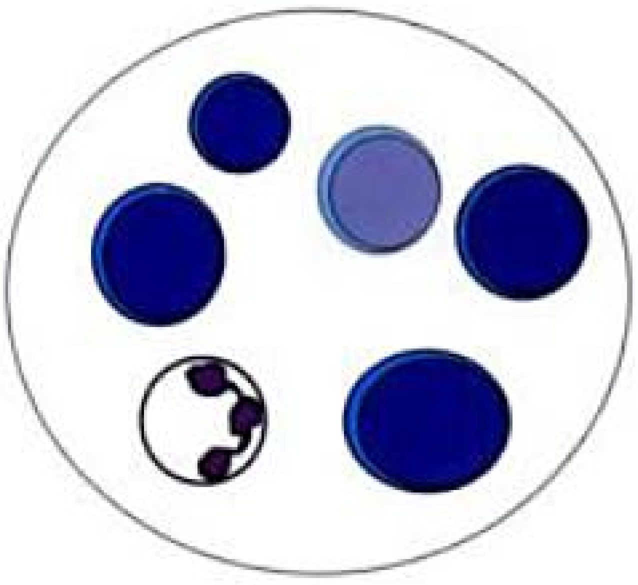
Lymphocytic/plasmacytic inflammation

Appearance (×1000)

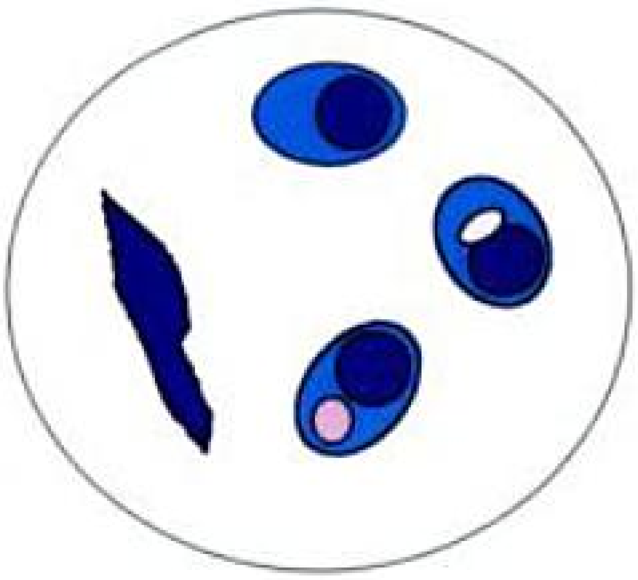
Interpretation



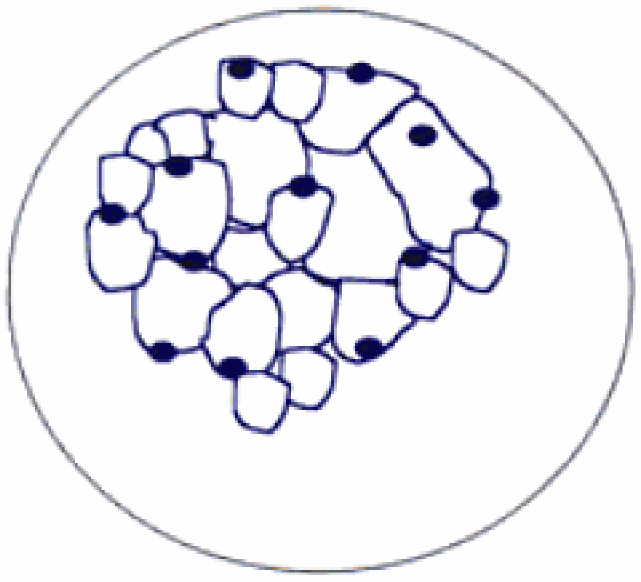
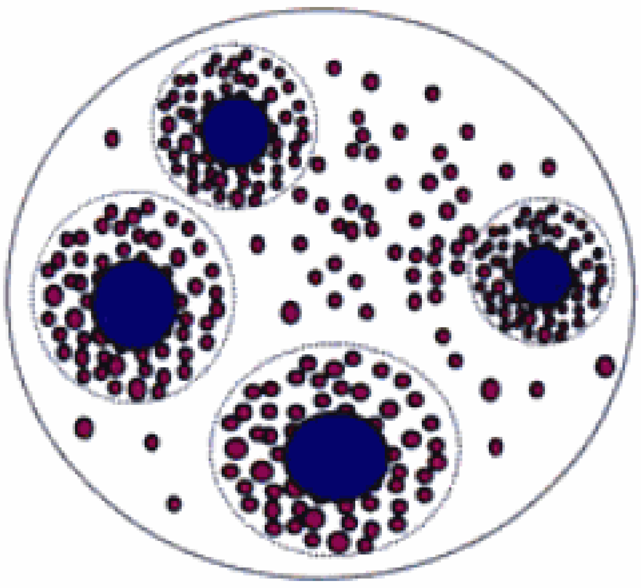
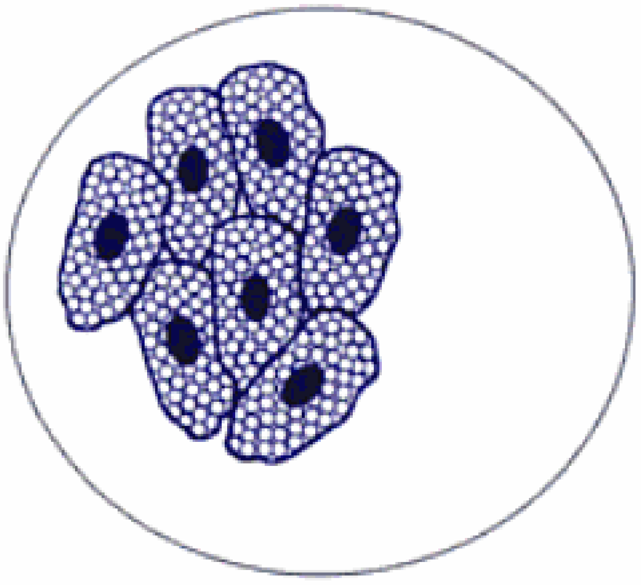
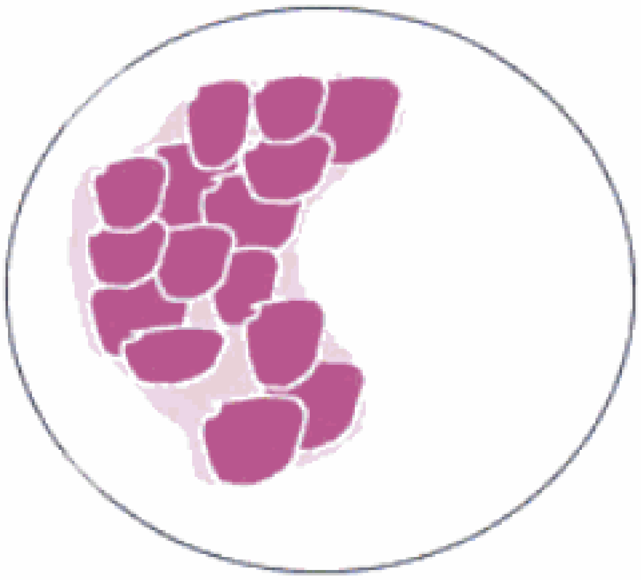
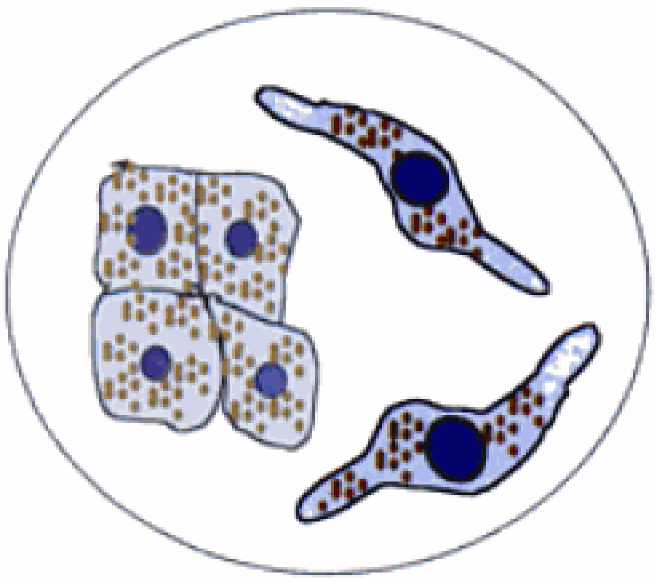
The presence of lymphocytes in skin cytology is much less common than neutrophils, macrophages or eosinophils. Lymphocytes appear as small, densely staining cells with little or no visible cytoplasm. It is important when assessing lymphocytes to be aware of scale. It is usually possible to find a neutrophil somewhere on the slide and this can be used as a size marker. Lymphocytes are smaller than neutrophils. Occasional lymphocytes can be found in any inflammatory skin disease.

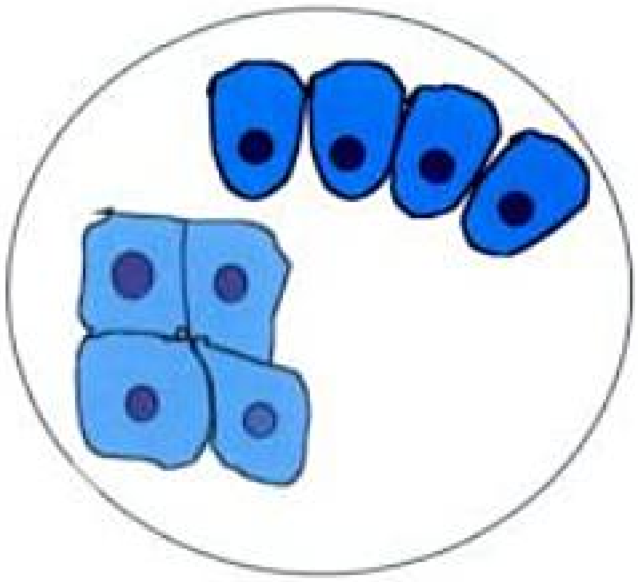


The presence of large numbers of lymphocytes with varying shapes and sizes (especially if some are larger than neutrophils) should prompt a search for cutaneous lymphoma. Biopsy would be indicated.

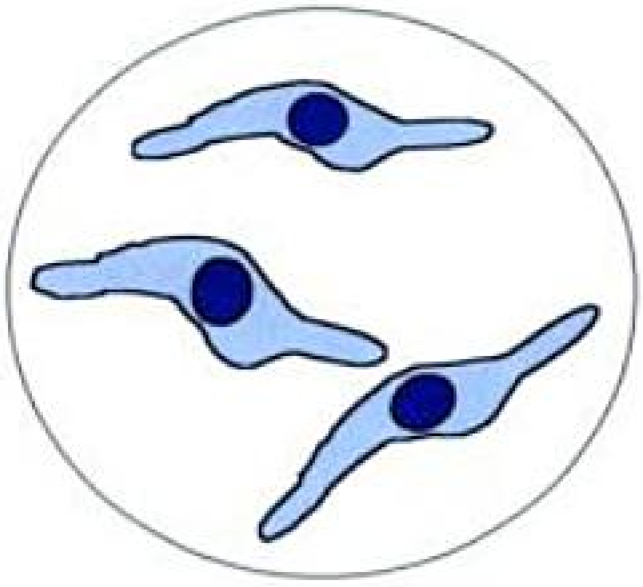


When stimulated to produce immunoglobulins, lymphocytes become plasma cells. These are oval in shape with an eccentrically placed nucleus (upper cell). A peri-nuclear clear zone representing the Golgi apparatus may be seen in some cells (middle cell). Some plasma cells may contain a pink staining body within the cytoplasm (lower cell). These cells are known as Mott cells and contain synthesised immunoglobulin. The presence of occasional plasma cells merely indicates that an immune response is occurring and is not specific for any skin disease.

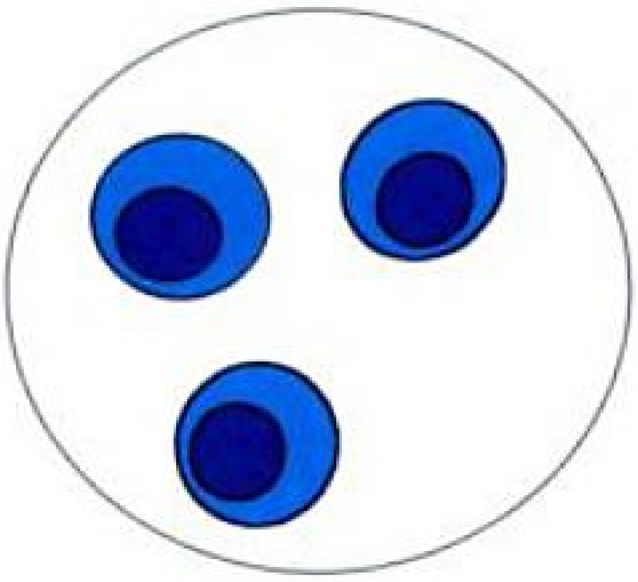
Appearance (×1000)	Interpretation
	Fine needle aspiration of this mass has revealed a monomorphic population of a few large cells with a foamy "soap-bubble" appearance. The cells have a small, eccentrically placed nucleus and there is no observable cytoplasm. This appearance is diagnostic of a lipoma.
	This field shows a monomorphic population of mast cells (large mononuclear cells with a central nucleus and cytoplasm that is packed with purple granules). This appearance is diagnostic of a mast cell tumour. Some of the cells have degranulated resulting in free granules being visible in the background. These granules should not be confused with bacteria.
	This fine needle aspirate has revealed a monomorphic population of large mononuclear cells. The cells have a central nucleus and the cytoplasm is full of foamy vacuoles. This is the characteristic appearance of sebaceous gland cells and would typify a nodule of sebaceous hyperplasia or a sebaceous adenoma.
	This field shows a mixture of pale, basophilic, non-nucleated cells amid a background of proteinaceous debris. This appearance is consistent with a non-infected follicular cyst.
	This field contains a number of cells with varying morphology, ranging from epithelioid to spindle cell. All the cells contain a large number of melanin granules. This appearance would be consistent with a melanoma.



This fine needle aspirate has revealed a monomorphic population of cells that have remained as sheets or clumps. The cells are joined together and have adhesions between them. This is most typical of an epithelial tumour such as a basal cell tumour, squamous cell carcinoma, hair follicle tumour or sweat gland tumour. Biopsy would be indicated.



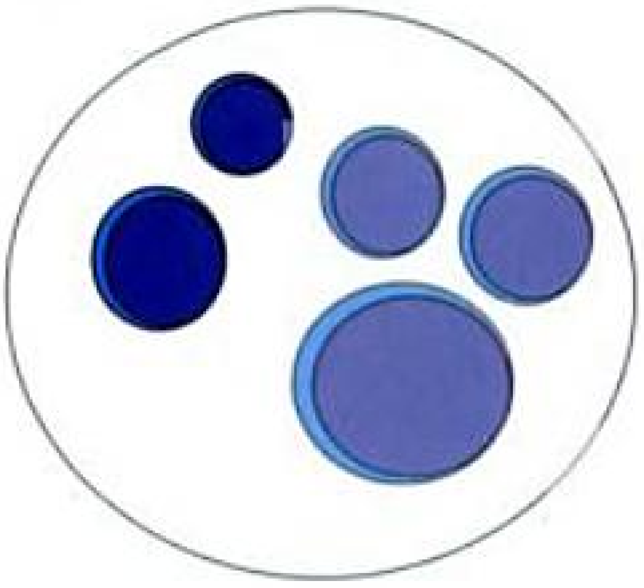
In this sample, there is a monomorphic population of cells with long extensions of poorly stained cytoplasm. The cells are not closely attached to each other and do not form sheets. These features are typical of spindle cell (mesenchymal) tumours such as fibromas, haemangiomas, haemangiopericytomas and sarcomas. Biopsy would be indicated.

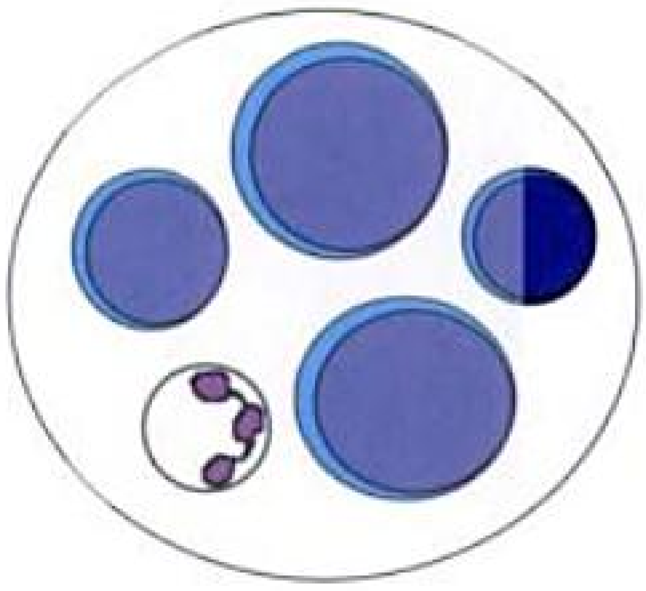


This field contains a monomorphic population of round cells which are not attached to each other by intercellular adhesions. This appearance is most consistent with a round cell tumour such as a histiocytoma, lymphoma, plasmacytoma or transmissible venereal tumour. Biopsy would be indicated for the last three conditions.

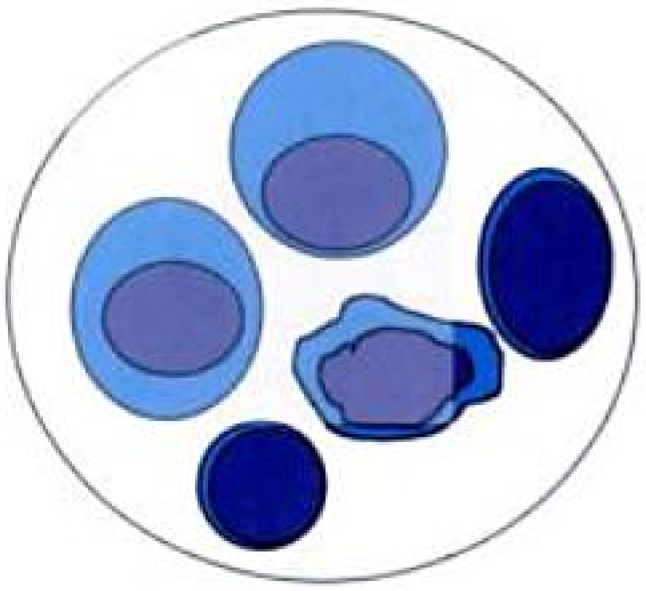
Criteria for malignancy

The following cytological changes increase the likelihood of a tumour being malignant.

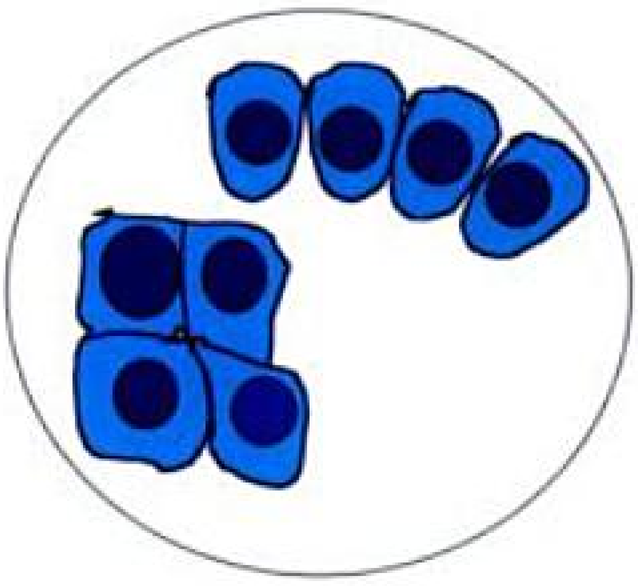
Appearance (×1000)	Interpretation
	Anisocytosis – the cells are different sizes.



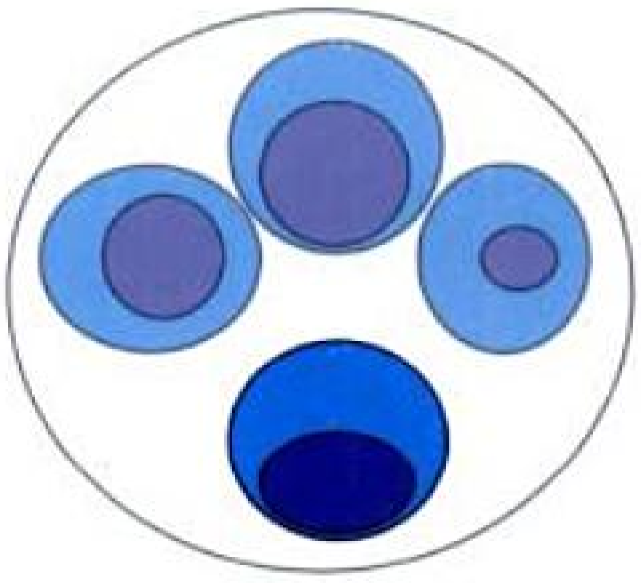
Macrocytosis – the cells are bigger than normal.



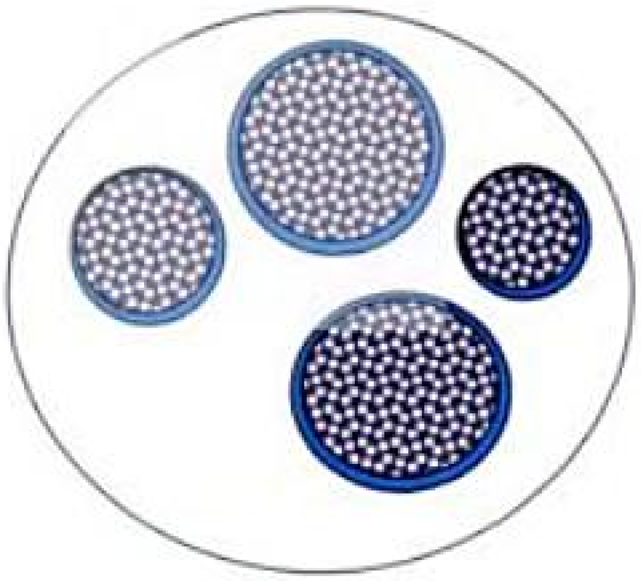
Pleomorphism – the cells are different shapes and sizes.



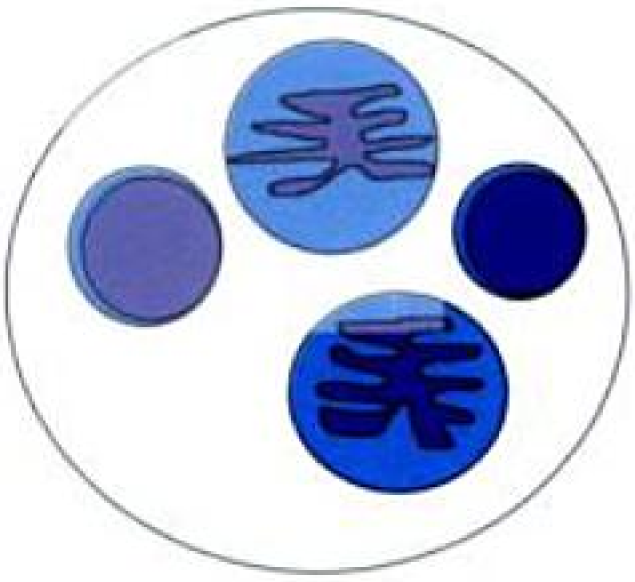
The nuclear:cytoplasmic ratio is increased.



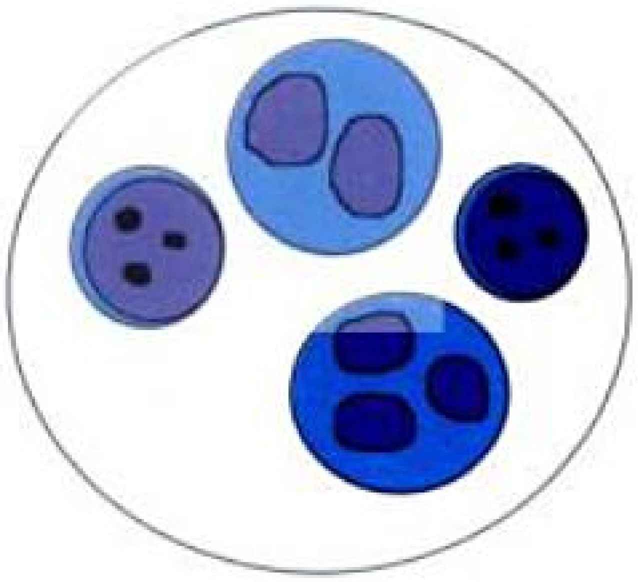
There is variation in nuclear size.



The nuclei have a course chromatin pattern.



Mitotic figures are present.



The cells have multiple nuclei or nucleoli.

Trichography

A trichogram is a simple diagnostic test in which a sample of hairs is examined under the microscope.

Technique	Simple
Interpretation	Requires a small amount of practice and experience
Sensitivity	50–90% (depends on disease and clinician's experience)
Specificity	90–100%

Indications

Should be performed in any animal presenting with alopecia.

May be helpful in animals presenting with pruritus or scaling.

May provide diagnostic information in cases of:

- Demodicosis
- Pediculosis
- Cheyletiellosis
- Dermatophytosis
- Feline symmetrical alopecia
- Endocrine alopecias
- Colour dilution alopecia
- Trichorrhexis nodosa.

Technique

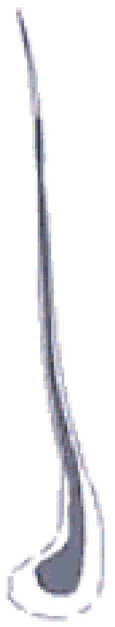
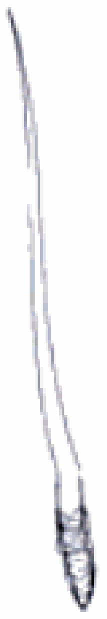
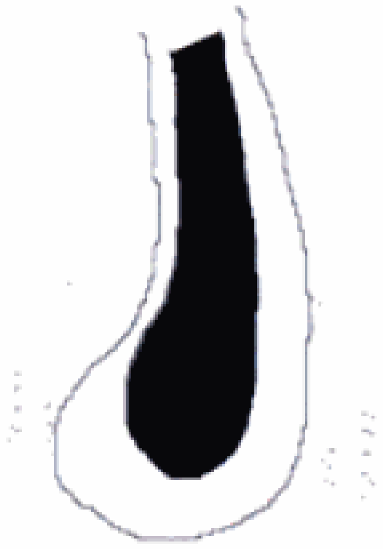
1. Grasp a number of hairs as close to the skin surface as possible using a pair of artery or epilation forceps. Ideally, the ends of the artery forceps should be covered in rubber sleeves to reduce trauma to the hair shafts.

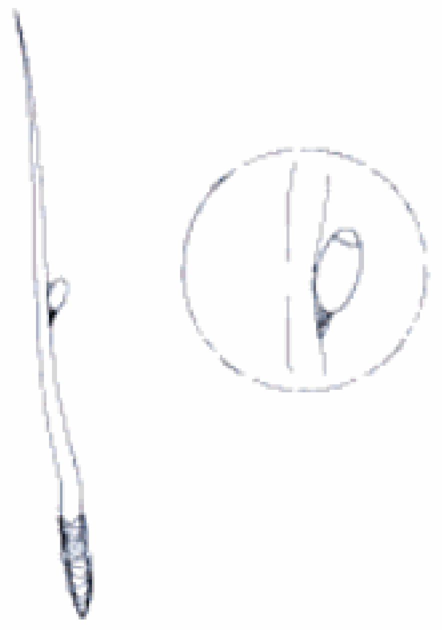
2. Pluck the hairs sharply.
3. Lay the hairs onto the surface of a drop of mineral oil on a slide. Make sure that the hairs are all oriented in the same direction.
4. Cover with a cover slip.
5. Examine under the 4×, 10× and 40× objectives of the microscope.

Examination

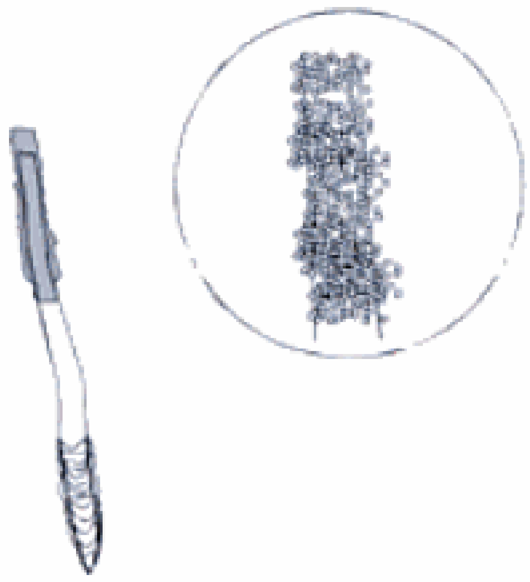
The hairs should first be examined under low power to assess the numbers, orientation and any obvious abnormalities that require closer scrutiny. The hairs should then be examined under higher power, starting at the base of the hair and working along the shafts to the points. The hairs should be assessed for the presence of anagen or telogen bulbs; peri-bulbar demodex mites; a smooth shaft; eggs attached to the shafts; debris or fungal spores attached to the shaft; any breakages in the shafts; the size and distribution of melanin granules within the shaft; and the presence of a tapering point.

The following figures aid in the interpretation of trichograms.

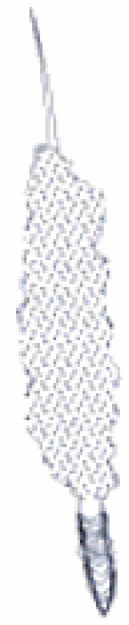
Appearance	Interpretation
	Normal anagen hair with smooth edges, a pigmented medulla, a smooth tapering point and an anagen bulb. Anagen bulbs are present on actively growing hairs and are smooth and rounded, often have a pigmented centre and commonly fold around the side of the hair shaft when plucked. The presence of large numbers of anagen hairs indicates that active hair growth is occurring in the area from which the sample was obtained.
	Normal telogen hair with smooth edges, a smooth tapering point and a telogen bulb. Telogen bulbs are present on inactive hairs and are spear-shaped, rough and lacking in pigment. If all the hairs in the trichogram have this appearance, it suggests that active hair growth is not occurring in the area from which the sample was obtained. This may result from endocrine diseases, telogen defluxion, and cyclic flank alopecia.
	The presence of demodex mites attached to the hair bulbs is diagnostic for demodicosis.



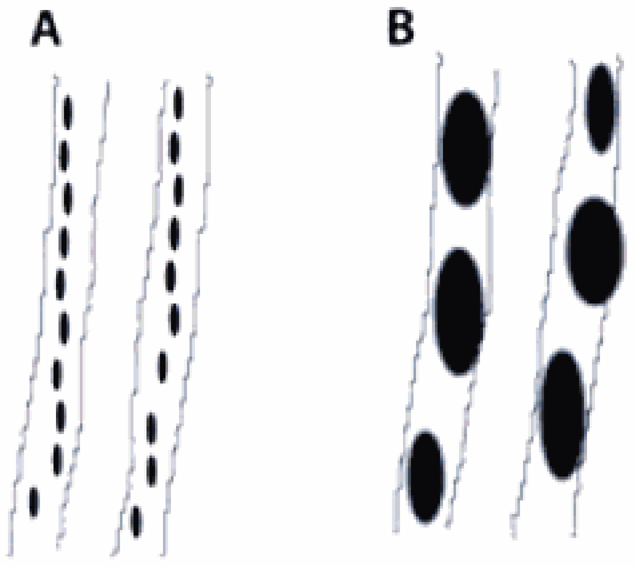
Hair shaft with an attached egg. This appearance is diagnostic for either cheyletiellosis or lice. Lice eggs are larger (visible to the naked eye) and attached to the hair shaft along their entire length.



Hair with a fractured shaft ensheathed in a mass of debris. On high power, the debris is composed of large numbers of ectothrix spores surrounding the hair shaft. This appearance is diagnostic for dermatophytosis.

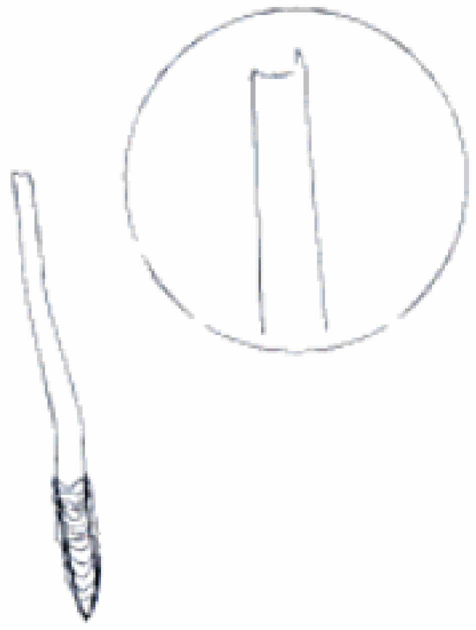


Normal hair shaft ensheathed in a mass of keratinaceous debris. This is a follicular cast and can be seen in cases of demodicosis, dermatophytosis, endocrine alopecias and cornification defects.

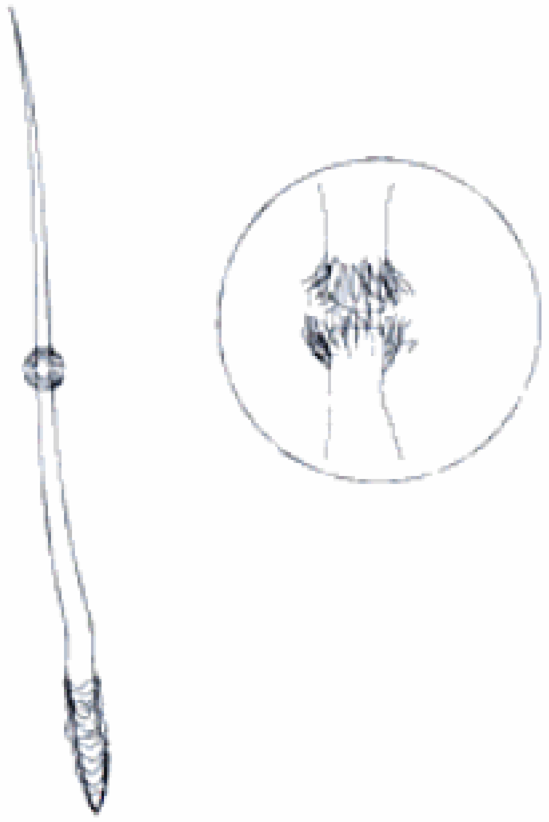


A: Normal melanin granules in the cortex of a hair shaft (micromelanosomes).

B: Abnormally large melanosomes seen in the cortex of a hair shaft (macromelanosomes). These are seen in animals with colour dilute coats and are seen in cases of colour dilution alopecia and black hair follicular dysplasia.



Hair with a fractured shaft. The hair does not taper to a point, but has an abrupt fragmented end. If most of the hairs have this appearance, then self-inflicted hair loss has occurred. This is seen in any pruritic skin disease but especially feline symmetrical alopecia.



Hair with a circumscribed bead-like abnormality in the mid-shaft. On high power magnification, the shaft looks frayed with the appearance of two brushes pushed together. This is indicative of trichorrhexis nodosa, a rare disease caused by trauma to the hair shaft (usually as a result of excessive brushing or shampooing) superimposed on an inherent weakness.

Tests for dermatophytosis

Four diagnostic tests are useful in the diagnosis of dermatophytosis: Wood's lamp examination, microscopic examination of hairs (trichography), fungal culture and skin biopsy.

Wood's lamp examination

A simple diagnostic test in which an ultraviolet lamp is used to detect fluorescing hairs. The technique is only useful for dermatophytes that produce fluorescent metabolites (*Microsporum canis* is the only common one).

Technique	Simple
Interpretation	Simple
Specificity	90%
Sensitivity	≈ 50% (test may fail to detect the dermatophyte infection)

Indications

Should be performed in any animal presenting with lesions consistent with dermatophytosis.

Technique

1. Allow the lamp to warm up for 5 minutes before using.
2. Examine the animal in a darkened room. Allow your eyes to become acclimatised to the dark before starting.
3. Examine the whole of the hair coat (not just the lesions).

4. A positive result is an apple-green fluorescence of the hair shafts. A blue-purple iridescence is often seen associated with dust particles.
5. If a positive result is obtained, confirm the finding with direct microscopy and/or a fungal culture.

Microscopic examination of hairs

Direct examination of hairs for the presence of fungi is simply a form of trichography (see also Trichography page 176).

Technique	Simple
Interpretation	Requires practice and experience
Specificity	100%
Sensitivity	≈ 50% (test may fail to detect the dermatophyte infection)

Indications

Should be performed in any animal presenting with lesions consistent with dermatophytosis.

Technique

1. Ideally, collect fluorescing hairs for microscopic examination. If not, collect broken, stubbly hairs from around the periphery of lesions.
2. Hairs should be plucked sharply using epilation or artery forceps.
3. Place the hairs on a glass slide in a drop of mineral oil or a clearing agent and cover with a cover slip. Although mineral oil is an acceptable mounting medium, clearing agents bleach the melanin content of hair shafts allowing fungal spores to be more easily seen.

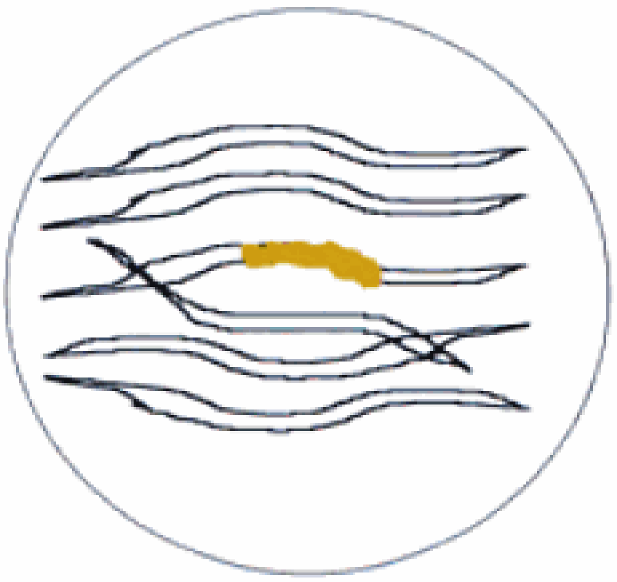
Suitable clearing agents include:

- 20% KOH – leave the slide to stand for 30 minutes or heat for 15 seconds.
- Two parts 20% KOH to one part blue-black Quink ink (this stains the spores and hyphae).
- Chlorphenolac (50 g chloral hydrate, 25 ml liquid phenol, 25 ml liquid lactic acid).

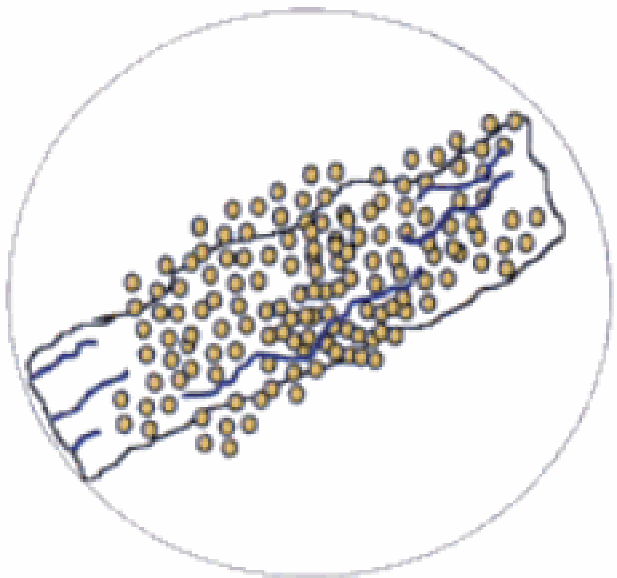
4. The examination and interpretation of the slide is shown in the figures below.

Appearance

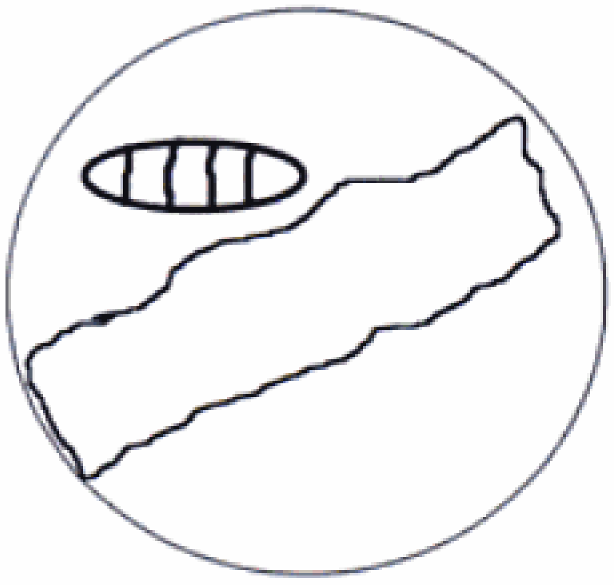
Interpretation



Start off at low power (×4 and ×10) and examine all the hairs. Look for those that seem to be covered in a sheath of debris.



Move up to high power (×400) and examine the affected hair. Look for a mass of spores covering the hair shaft or hyphae travelling along its length (stained blue in this example).



Structures such as this DO NOT form on the skin during a dermatophyte infection. This is a contaminant fungal spore picked up from the environment (e.g. *Alternaria*).

Dermatophyte culture

Both Wood's lamp examination and direct microscopic examination of hairs can be negative in cases of dermatophytosis (the sensitivity is not 100%). Hence, a negative result should never be used to rule out dermatophytosis. If the condition is still suspected a dermatophyte culture should be performed. Even if dermatophytosis has been diagnosed on direct microscopy, fungal culture is indicated to determine which species is present.

Technique	Simple
Interpretation	Requires some practice and experience
Specificity	100%
Sensitivity	95%

Indications

Should be performed in any animal presenting with lesions consistent with dermatophytosis.

Technique

1. The most reliable method of collecting a sample for fungal culture is to use the toothbrush technique. This involves brushing the hair coat and the lesions for 1–2 minutes with a new toothbrush. This picks up fungal spores, even if the level of infection is fairly low.
2. Samples can also be collected by plucking individual hairs. This is less sensitive because infected hairs might be missed. However, the sensitivity of the whole technique is increased by combining both hair pluckings and toothbrushings.
3. The bristles of the toothbrush and the collected hairs are used to inoculate the culture medium. This can either be performed by an external laboratory or undertaken in the practice laboratory. If an external laboratory is used, the whole toothbrush and hair sample should be sent away in a sealed envelope and jiffy bag.

The culture, identification and sensitivity testing of pathogenic bacteria and fungi involves the following steps:

1. Identification of appropriate lesions to sample
2. Collection of material for culture
3. Correct packaging and transport
4. Culture of the organism on the appropriate media
5. Tests to confirm the organism's identity
6. Sensitivity testing.

The first three steps are performed by the clinician. The remaining steps should be performed by a specialist microbiology laboratory that is capable of handling the suspected organisms and are not described in this chapter.

Bacterial culture

Techniques	Simple
Interpretation	Simple
Specificity	100%
Sensitivity	80–100% (test can occasionally fail to detect the infection)

Indications

Bacterial skin infections should be initially assessed on the basis of clinical lesions and cytology. In the case of canine superficial pyoderma or cat bite abscesses, this is all that is required to make a diagnosis and commence appropriate antibacterial therapy. As these conditions are the most common bacterial skin infections in the dog and cat, bacterial culture is rarely required in veterinary dermatology.

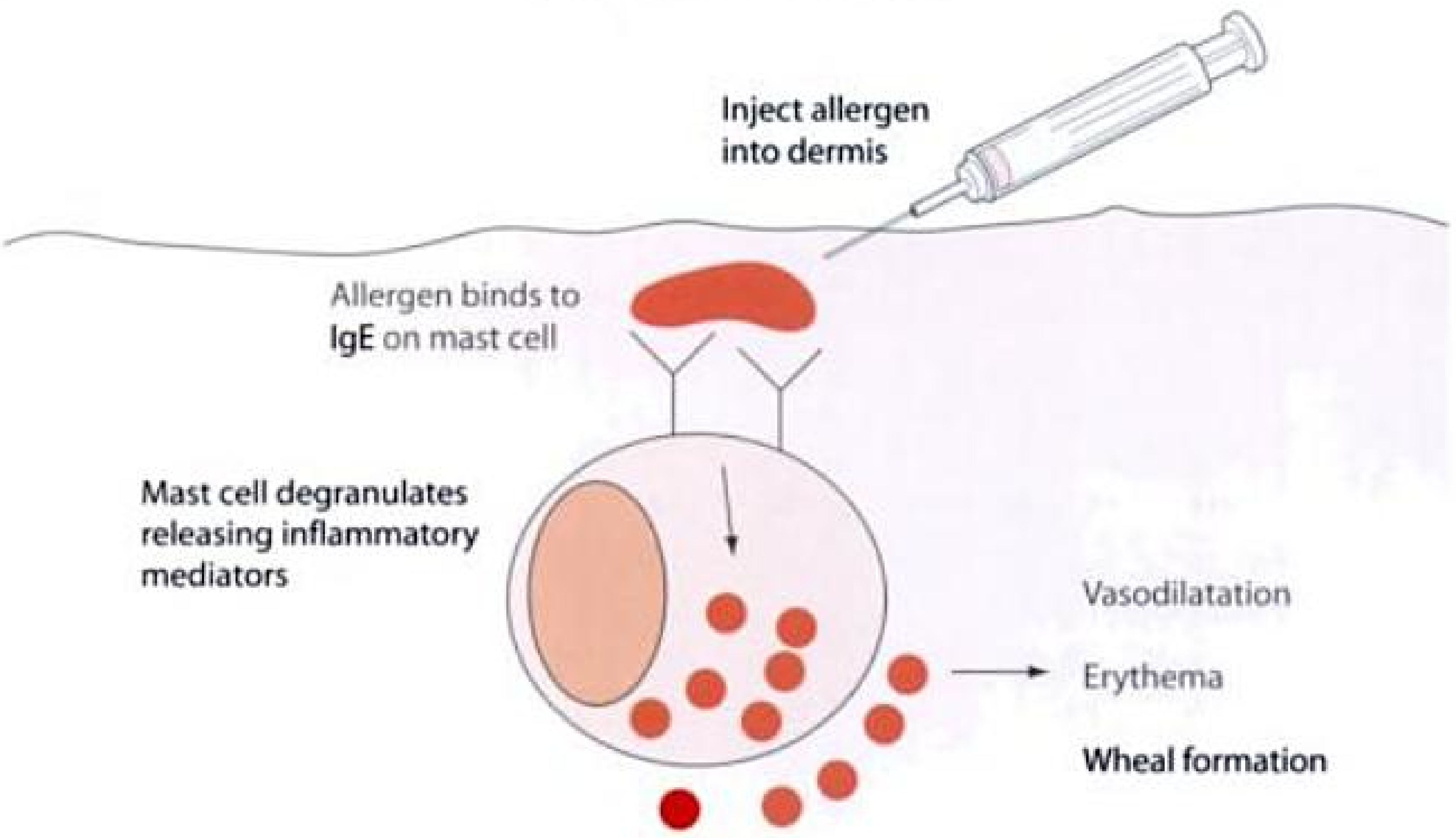
Bacterial culture should be performed in the following circumstances:

- In common skin infections that have failed to respond to appropriate treatment.
- In cases where draining tracts are present.
- In cases where nodular, granulomatous lesions are present.
- In cases where unusual bacteria are seen on cytology (rods, filaments).
- In cases of otitis externa where rods are seen on cytology.

Techniques

1. The clinician should first choose appropriate lesions for culture. These include intact pustules, draining tracts, nodules, and ear canal exudate. Sampling the periphery of epidermal collarettes is less satisfactory but is acceptable if no other lesion types are present. The surface of ulcers or erosions is not an acceptable site because these lesions are often contaminated by environmental bacteria.
2. Intact pustules should be opened with a sterile needle. Pustules should not be cleaned or disinfected prior to sampling because this can lead to negative results. A sterile culture swab should be touched against the purulent exudate and then placed in its sterile container. Culture swabs containing transport medium are preferred for dermatological use.
3. Draining tracts can be sampled by placing a sterile culture swab deep within the tract. Exudate that has been on the skin surface for any length of time should not be sampled because it is likely to be contaminated.

(a) Intradermal skin testing


Inject allergen into dermis
Allergen binds to IgE on mast cell
Mast cell degranulates releasing inflammatory mediators
Vasodilatation
Erythema
Wheal formation

(b) *In-vitro* measurement of allergen-specific IgE

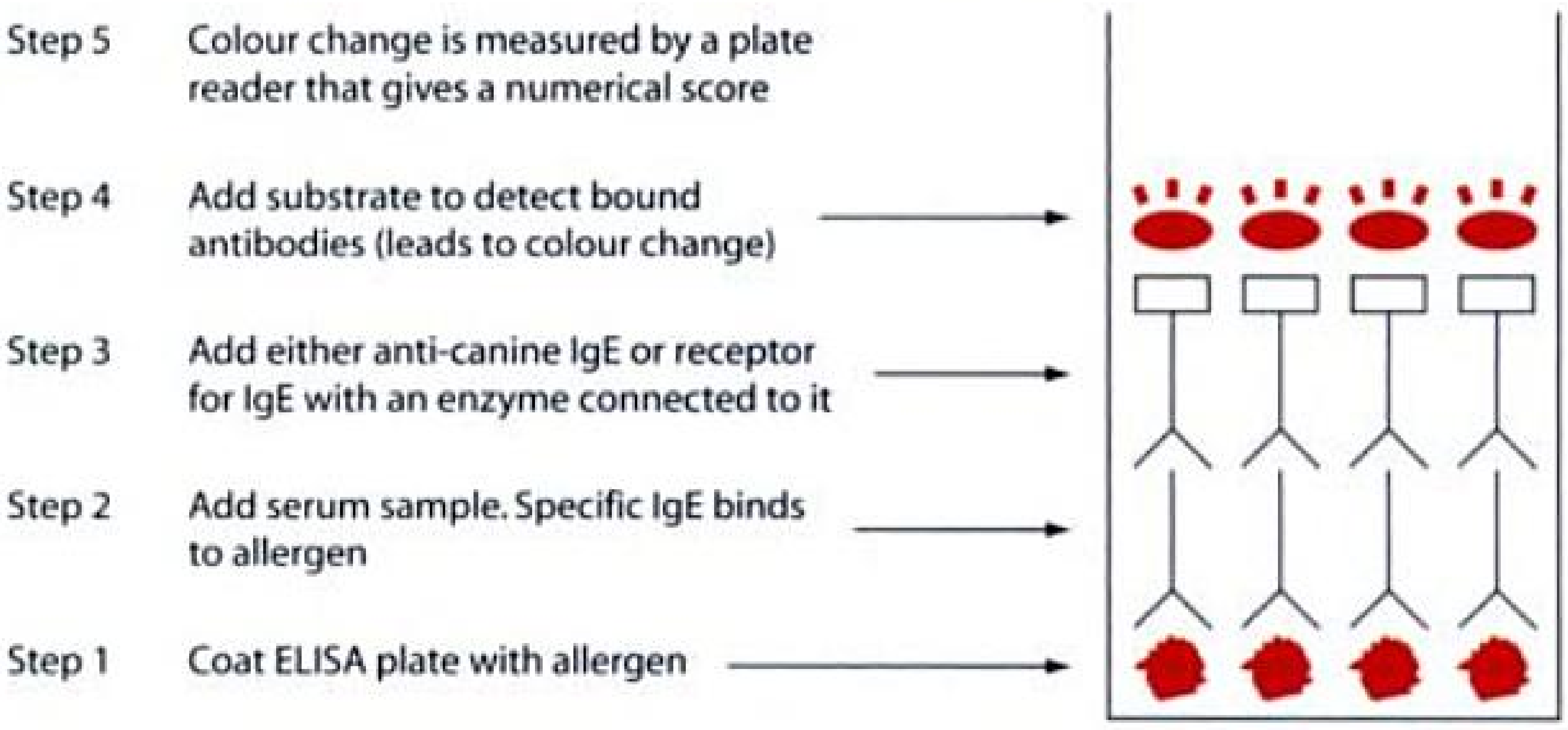

Step 5 Colour change is measured by a plate reader that gives a numerical score
Step 4 Add substrate to detect bound antibodies (leads to colour change)
Step 3 Add either anti-canine IgE or receptor for IgE with an enzyme connected to it
Step 2 Add serum sample. Specific IgE binds to allergen
Step 1 Coat ELISA plate with allergen

Figure 13.2 A comparison of allergy testing methods for atopic dermatitis

Positive results can be obtained with either test in clinically normal dogs and dogs with other skin diseases. The test result is only meaningful if the dog has clinical signs consistent with atopic dermatitis and all other pruritic diseases have been ruled out.

Intradermal skin test

The intradermal skin test is a method for demonstrating the presence of hypersensitivity to various environmental allergens based on skin reactivity.

Technique	Simple
Interpretation	Requires extensive practice and experience
Specificity	≈ 50–90% (specificity increases as other pruritic skin diseases are ruled out).
Sensitivity	≈ 90%

Intradermal skin testing is usually performed at referral centres or in practices in which there is a clinician with a specific interest in dermatology. The selection of allergens and interpretation of intradermal skin tests requires specialised advice and training. Allergen solutions are also expensive and it would not be cost effective to offer this service unless one or two tests per week were being performed.

Practitioners interested in performing this procedure should visit a specialist veterinary dermatologist, study for a further qualification in the discipline or undertake residency training. It is beyond the scope of this book to provide comprehensive details of skin testing procedures. The intention of this chapter is to provide veterinarians with sufficient background to the methodology to allow appropriate case selection and to be able to inform clients about the procedure.

Most veterinary dermatologists test for reactivity against the following antigens:

- House dust mite and storage mite antigens (*Dermatophagoides farinae, Dermatophagoides pteronyssinus, Acarus siro, Tyrophagus putrescantiae, Euroglyphus maynei*).
- Human and animal danders (derived from desquamated corneocytes).
- House dust (a mixture of dust mite antigens, human and animal danders, bacteria, fungal spores, and fibres).
- Pollens (from trees, weeds and grasses).
- Moulds (from the household or from crops).

The inclusion of regional allergens (pollens) in the testing kit is based on knowledge of the plants in a particular geographical location. In large countries such as the USA, there are distinct botanical zones that contain species of trees, grasses and weeds not found in other areas. In smaller countries such as the UK, this is not a major problem.

Indications

Indicated in animals with a history and clinical signs consistent with atopic dermatitis in which allergen immunotherapy is considered the treatment of choice. Should only be performed after other pruritic diseases have been ruled out or controlled (ectoparasites, pyoderma, *Malassezia* dermatitis, food allergy).

Technique (summarised in Figure 13.2)

1. The procedure is best performed with the animal under sedation in lateral recumbency. Xylazine (Rompun) at a dose of 0.5–0.75 mg/kg or medetomidine (Domitor) at a dose

of 10–20 µg/kg are the preferred sedatives. Acepromazine (ACP) is not acceptable because it reduces skin test reactivity.
2. A patch of fur is clipped from the lateral thorax (about 6″ × 4″(15 × 10 cm)).
3. The injection sites are marked using a black marker pen.
4. Approximately 0.05 ml of each antigen is injected intradermally along with the positive (histamine) and negative (saline) controls (the exact amount is not critical as long as all the injections are the same size).
5. The reactions are read 10–20 minutes later. These appear as wheals. The positive control is given a score of 4+ and the negative control a score of 0. Other reactions are subjectively graded between these values based on the diameter of the wheal, the degree of erythema and the height of the wheal.
6. In some cases, late phase reactions may occur at some sites 24–48 hours later. These appear as erythematous, indurated areas that may contain a papular eruption. The full significance of these reactions is currently unknown. Some dermatologists ask owners to observe for these reactions whereas others hospitalise the animal and reassess the skin test site themselves.

Interpretation

The skin test is interpreted by correlating the positive reactions with the patient's history. Clinically relevant reactions can then be used to choose allergens for specific immunotherapy.

In vitro measurement of allergen-specific IgE

In-vitro testing simply requires a blood sample to be taken and sent off to an appropriate laboratory. The serum is assayed for allergen-specific IgE and the results are reported as relative units (the higher the score, the higher the level of IgE).

Technique	Simple
Interpretation	Simple
Specificity	≈ 50–80% (specificity increases as other pruritic skin diseases are ruled out).
Sensitivity	≈ 90%

Indications

Indicated in animals with a history and clinical signs consistent with atopic dermatitis in which allergen immunotherapy is considered the treatment of choice. Should only be performed after other pruritic diseases have been ruled out or controlled (ectoparasites, pyoderma, *Malassezia* dermatitis, food allergy).

Technique (summarised in Figure 13.2)

1. A blood sample is taken and the serum sent off to a laboratory offering the service.
2. The diluted serum sample is added to a plate containing individual wells coated with specific antigens. The range of antigens is similar to that used for intradermal skin testing.
3. If there is any IgE in the serum that is specific for a particular antigen, it binds to it. The bound IgE is then detected by adding an enzyme-linked reagent that can bind to IgE. This is either a monoclonal antibody or a receptor for IgE molecules.
4. A substrate is added that changes colour when it contacts the enzyme attached to the IgE reagent. The degree of colour change is proportional to the amount of IgE that is bound.

Tests to investigate food allergy

Unlike many diseases, there are no currently available laboratory or clinical tests that can be used to diagnose food allergy in dogs and cats. Both intra-dermal tests and *in-vitro* measurement of allergen-specific IgE are regarded as unreliable indicators of food sensitivities and can not, at present, be recommended for use. Food allergy can only be diagnosed by properly conducted dietary trials.

Technique	Preparing home cooked diets is labour intensive, feeding commercial diets is simple.
Interpretation	Complex, with numerous pitfalls
Specificity	0–100% (depends on rigour of interpretation)
Sensitivity	0–100% (depends on diet chosen, owner compliance)

Indications

Should be performed on any dog or cat with a chronic pruritic skin disease after ruling out ectoparasites and infectious causes.

Technique

In many textbooks and publications, the diagnosis of food allergy is described as a simple matter in which the animal is placed on a restricted diet trial to determine if the skin condition resolves. If it does, the animal is placed back onto its original diet and observed for signs of a recurrence. For many clinicians, this would be diagnostic for food allergy. Although this is the basic premise on which food allergies are diagnosed, there are a number of practical considerations that need to be addressed if any meaningful information is to be derived from this commonly recommended procedure. Before commencing a dietary trial, the clinician must know:

1. Whether to recommend a home-cooked or commercially available diet.
2. How to prepare and feed a home-cooked diet.
3. What problems might occur with home-cooked diet trials.
4. How long diet trials should last.
5. How to assess the response to dietary trials.

Deciding on a home-cooked or commercially available restricted diet

The first dilemma faced by the clinician is to decide whether to recommend a home-cooked diet trial or use a commercially available limited ingredient diet. Most veterinary dermatologists would agree that home-cooked diets are preferable. The rationale for this is based on limited scientific evidence that some dogs and cats (about 20%) can be maintained on a home-cooked diet but show a recurrence of signs when fed a commercially available diet containing the same ingredients. This is thought to be due to the additives that are present in commercial diets (colourings, flavourings, preservatives, vitamins, minerals) that are used to ensure the diets are nutritionally complete, palatable and resistant to spoilage. An alternative possibility is that food sources are antigenically changed during the manufacturing process. Although neither of these possibilities have been definitively proven, a home-cooked diet trial is still regarded as the optimal method to definitively rule out food allergy. This is because if there is no response to the diet (as occurs in the majority of cases), there is no residual element of doubt that the animal could be reacting to an additional dietary component.

However, home-cooked diet trials are labour intensive and can cause problems with owner compliance. They are also not suitable for growing animals or animals already on prescription diets for other purposes. Therefore, if a home-cooked diet trial is not possible, a commercially available limited ingredient diet trial is preferable to not performing a diet trial at all. Lists of currently available commercial limited ingredient diets are provided in Chapter 14 under "Treatment of food allergy".

Preparation and feeding of home-cooked restricted diets

The usual recommendation for formulating home cooked diets is to restrict the animal to one single protein source, one single carbohydrate source and water. The choice of these ingredients should be based on a detailed analysis of the animal's previous dietary history. Typical protein sources include fish (coley, cod, haddock, tuna), pork, rabbit, venison, turkey or lamb. Owners can purchase these foods fresh from a butcher or fishmonger, or can obtain them frozen from supermarkets or pet superstores. Beef and chicken are usually avoided because these are common ingredients in commercial pet foods. Typical carbohydrate sources include potato, rice or pasta.

Although the individual ingredients vary in a particular diet, the preparation is fairly standard. The meat can be boiled, baked or microwaved and the carbohydrate can be boiled as usual. Excess fat should be removed from the meat as this may lead to diarrhoea. The meat and carbohydrate can then be mixed together in a blender or food processer. This provides a mixture that the animal is unable to separate. The ratio of the protein source to the carbohydrate should be about 1:2. The mixed food can then be weighed, placed in bags and frozen until required.

The preparation of home-cooked diets can be a very time-consuming process and requires quite large cooking utensils. For small dogs (5–10 kg) and cats, it is just about possible to prepare the food on a weekly basis using the typical pans and bowls that are found in an average household. This is likely to involve a preparation time of about 2 hours. The food can then be stored in the freezer until required. However, for larger dogs, it is likely that the food will need to be prepared on a more regular basis due to limitations of utensil size or freezer space. This increases the weekly preparation time to between 4 and 6 hours. For large dogs, the cost of preparing home-cooked restricted diet trials can be considerable. This can be minimised by advising the owner to purchase frozen meat minces and frozen fish fillets that are available in most large supermarkets.

Owners often ask how much of the home-cooked diet should be fed. In general terms, home-cooked diets are nutritionally incomplete and underfeeding is more of a problem than overfeeding. To ensure adequate intake, owners can be advised in one of the following ways:

- Feed an amount according to the animal's body weight (see Table 13.2)
- Start with a quantity about half as much again as the animal's normal commercial diet. Further increases can be made if the animal appears constantly hungry or begins to lose weight. It is advisable to weigh animals on a weekly basis throughout a food trial in order to monitor body weight.
- Some dermatologists merely recommend that the diet is offered *ad libitum*. However, with home-cooked diets, this is not as easy as pouring extra biscuits out of a bag and it can also lead to wastage.

Which of these methods is advised depends on the clinician and client's preference.

Table 13.2 gives approximate guidelines for feeding home cooked diets. These amounts may need to be modified in light of the breed, activity level, climate, appetite and weight changes. Amounts represent daily intake and indicate cooked weights.

Taurine deficiency in cats is a theoretical concern but most veterinary dermatologists do not provide this supplement during the duration of the trial. However, if a home-cooked diet is to be used beyond the period of the trial, it is essential that it is balanced with additional ingredients (see Chapter 14). In addition, home cooked-diets should not be fed to growing animals and those with special nutritional needs.

Recommended duration of restricted diet trials

The length of time that a restricted diet needs to be fed is a controversial area in veterinary dermatology. In many older publications and standard textbooks, a 3-week trial was advocated. This was based empirically on anecdotal evidence, clinicians' own experiences and a general feeling that the allergic reaction should subside within this time after the offending allergens were removed. However, more recent studies have suggested that the duration of the restricted diet trial should be extended to 10 weeks in some cases. These publications were based on the observation that up to 70% of dogs and cats showed no response to the diet trial at 3 weeks but showed complete or partial resolution of clinical signs by 6 or 10 weeks. Because of the many problems associated with assessing the response to home-cooked food trials (see below), some dermatologists have questioned the validity of this data. Furthermore, other recent studies have contradicted these findings, suggesting that a clinical response to diet trials is seen within 3 or 4 weeks. It is therefore difficult to make firm recommendations when the literature is replete with such conflicting data. Clearly, the diet can be challenged as soon as the animal shows a response, even if this is within the first 3 weeks. If there has been no response by this time, many dermatologists continue the trial for a further 3 weeks. Whether the trial should be continued beyond this time depends on the compliance of the owner and the index of suspicion of the clinician.

Assessing the response to food trials

There are many problems and pitfalls in assessing the response to dietary trials. Although many animals appear to show a response to restricted diets, this does not confirm a diagnosis of food allergy. The clinician needs to take into account all the following factors before accepting that the diet is playing any role in an animal's skin disease.

Owner compliance

As a result of the practical difficulties associated with formulating and feeding home-cooked restricted diet trials, there can be major problems with owner compliance. Some owners admit beforehand that it will not be possible to perform the diet as recommended. Although this poses further diagnostic dilemmas, it may be an honest reflection of the owners capabilities and financial situation. Of greater concern are the many clients that agree to perform the food trial in the consulting room, but then fail to carry it out properly because of the practical problems they encounter when attempting it. Even after receiving the client's assurance that the diet has been correctly performed, there is no way of determining, based on the owner's history alone, that this is the case. The veterinarian must therefore accept that some cases of food allergy may remain undiagnosed despite extensive dietary trials.

Failure to re-challenge

If an animal appears to have made a spectacular recovery during a restricted diet trial, some owners are reluctant to put their pet back onto its original diet. Whilst it is tempting to share this view, it is important to insist that the animal is challenged with its previous food. The main reason for this is that, as with any therapeutic trial in veterinary dermatology, there can be a significant placebo effect (in up to 20% of cases). Hence, a restricted diet could be continued for prolonged periods of time without being of any benefit to the patient. This situation can be seen in cases that undergo spontaneous resolution or those that receive some other concurrent treatment at the time of the trial (anti-parasiticides or antimicrobial agents). The original diet should therefore be gradually introduced over a 3 to 4 day period and the animal observed for signs of a relapse over the next 7 to 10 days.

Partial or coincidental responses

Partial responses to diet trials may occur in animals that have atopic dermatitis and food allergy at the same time, but can also occur when another disease undergoes spontaneous fluctuations in severity (waxing and waning). Based on the currently accepted criteria for diagnosis of food allergies (response whilst on the restricted diet, relapse on challenge, followed by a further response on re-introduction of the restricted diet), it is still possible to make an erroneous diagnosis of food allergy based solely on coincidence. The only way to overcome this problem is to repeat the cycle on a number of occasions until both the veterinarian and the client are totally convinced that the diet is a major factor. However, repeated cycling is time consuming and causes further difficulties with client compliance. To date, no series of cases reported in the literature has used this approach and demonstrated convincing long-term follow-up data to fully substantiate the diagnosis of food allergy in all cases.

Influence of secondary bacterial and yeast infections

A major problem encountered when assessing dietary trials is the development and control of secondary infections such as staphylococcal pyoderma and *Malassezia* dermatitis. These infections are common in dogs with cutaneous hypersensitivities and specific treatment for them is often prescribed at the same time as the restricted diet trial. When the animal returns for re-examination, there may have been a dramatic response and a significant or complete reduction in pruritus. It is critical when faced with this situation that the antimicrobial therapies are continued throughout the challenge phase of the diet trial. If they are not, it is likely that the secondary infections will recur and give the impression that the dog has relapsed on reintroduction of the normal diet, again leading to an erroneous diagnosis of food allergy.

Concurrent anti-pruritic therapy

The use of anti-pruritic therapy such as glucocorticoids at the same time as the food trial can greatly complicate the assessment of the patient's progress. It is critical, therefore, that glucocorticoids are used only when absolutely necessary (for example, in dogs or cats that are so pruritic that they are self-mutilating themselves). In this case, the glucocorticoids must be given orally and for the shortest possible time (1–3 weeks). They must then be withdrawn completely so that the effects of the diet trial can be assessed.

Provocative testing

If, despite all the difficulties outlined above, the clinician makes a definitive diagnosis of food allergy, it is desirable to determine the specific antigens the animal is reacting to. This can be achieved by provocative testing. This is a series of sequential dietary challenges in which the animal is fed additional single protein and carbohydrate sources. To begin with, the animal is placed on the restricted diet until maximal improvement has been noted. Then, an additional single protein or carbohydrate source is added to the diet for 7 to 10 days. If there is no relapse, a second protein or carbohydrate source is added instead of the first one for a further 7 to 10 days. This cycle is repeated until the animal has been exposed to beef, chicken, pork, lamb, turkey, fish, egg, milk, bread, potato and rice. If, at any stage, the animal has a relapse, it must be placed back onto the restricted diet until maximal improvement has occurred again. The sequential challenge can then proceed in order to determine if any other sensitivities are present. This process provides the clinician and owner with information that allows the long-term dietary management of the animal to be accurately planned. However, it takes a highly committed and motivated owner to follow these guidelines correctly and return meaningful information. Many clients are simply happy to maintain the restricted diet or attempt to find a commercially available diet that the animal can tolerate.

Details on the long-term management of confirmed cases of food allergy are provided in Chapter 14.

Tests to investigate contact dermatitis

Four diagnostic tests can be used to investigate suspected contact dermatitis:

- Removal of suspected causes of contact irritation
- Environmental restriction
- Open patch testing
- Closed patch testing

Removal of suspected causes of contact irritation

A method for demonstrating contact dermatitis by removing something that is contacting the skin in the affected area.

Technique	Simple
Interpretation	Simple
Specificity	50–100% (removal of suspected cause may lead to coincidental improvement)
Sensitivity	100%

Indications

Should only be performed if the clinician strongly suspects that the skin lesions are being caused by a specific item that contacts the skin. This could be a collar, plastic food bowl, topical medication, shampoo, or floor disinfectant.

Technique and interpretation

1. The suspected item or substance is removed or not used. Complete resolution of the skin lesions suggests that the item or substance was responsible.
2. If the lesions recur following reintroduction of the item or substance, the diagnosis can be confirmed.

Environmental restriction

A method for investigating contact dermatitis by restricting an animal's access to certain environments.

can be applied as their normal formulations. Powders can be mixed with petroleum jelly. Floor cleaners and disinfectants can be applied at their normal working dilutions.
4. The sites are monitored daily for signs of erythema, oedema and pruritus.
5. Suspected positive reactions should be biopsied.

Interpretation

Open patch testing has not been standardised and there are no suitable positive or negative controls. Hence, it is not known what concentrations of substances should be applied. The applied substances can lead to irritant reactions that are not relevant to the animal's skin disease. Hence the reactions can be totally non-specific. Furthermore, if the concentration of applied substance is too low, a false negative result may occur indicating poor sensitivity. In view of these problems, open patch testing cannot be currently recommended.

Closed patch testing

A method for investigating contact dermatitis by applying certain items or substances to the skin.

Technique	Relatively difficult and infrequently performed
Interpretation	Very difficult, with numerous pitfalls
Specificity	0–100% (an irritant reaction may occur to something that is not the cause of the animal's dermatosis).
Sensitivity	0–100% (the appropriate substance may not be applied)

Indications

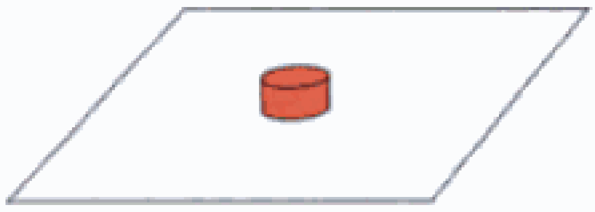
Should only be performed in cases with a history and lesions consistent with contact dermatitis in which a tentative diagnosis has been made by environmental restriction.

Technique

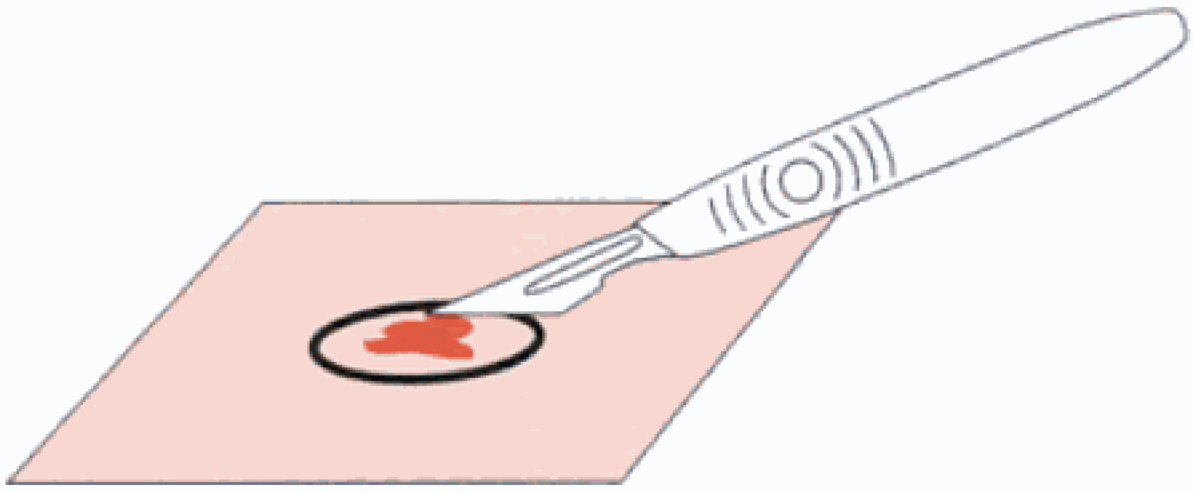
1. A patch of skin is clipped over the lateral thorax.
2. Suspected substances are applied either directly to the skin (solid material like carpets) or in small metallic cups known as Finn chambers (materials suspended in petroleum jelly or other vehicles). Commercially available patch testing kits are used in humans and contain a wide variety of potentially allergenic substances suspended in appropriate vehicles. These kits have not been validated for use in dogs and their specificity and sensitivity are not known.
3. The materials and Finn chambers are attached to the skin using adhesive tape and covered with a body bandage to prevent self-trauma.
4. The animal should be hospitalised to prevent self-trauma. The test materials are removed after 48 hours and the test sites monitored for the next 2 to 4 days. Positive reactions are manifested by areas of papular erythema and induration restricted to the sites that a particular material was applied.
5. Suspected positive reactions should be biopsied. Ideally, the offending substance should also be tested on a normal animal to ensure that it is not irritant.

Interpretation

Interpretation of patch testing is notoriously difficult and it can be plagued by false positive and false negative reactions. The body bandage can be dislodged by the animal and the test sites can be traumatised. The appropriate substances may not be applied, or if they are, in

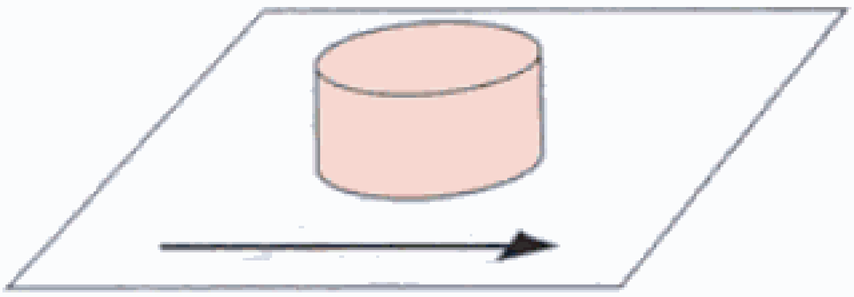


5. The biopsy should be gently cleaned by rolling it on a swab to remove artefactual haemorrhage. The biopsy is then placed on a square of cardboard, dermis side down. This is critical if the biopsy is to be correctly oriented (see below). The biopsy site should then be closed using a simple interrupted or cruciate suture.

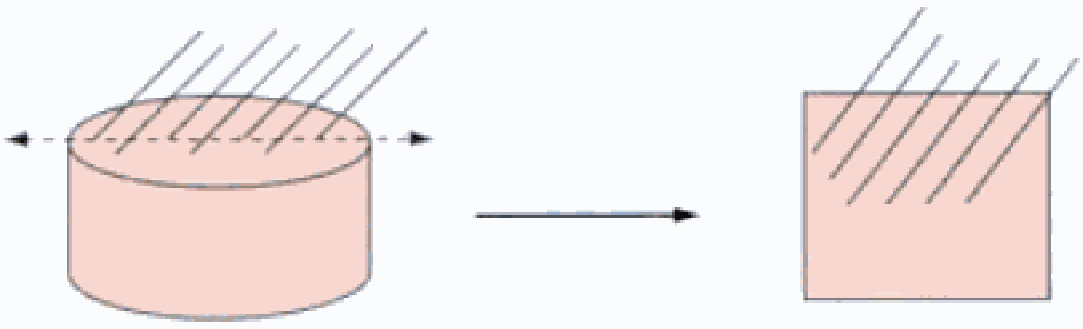


6. If larger or deeper biopsies are required (such as for lump removal or collection of material from the subcutis for tissue culture), a wedge or ellipse of tissue can be removed using a normal scalpel blade.

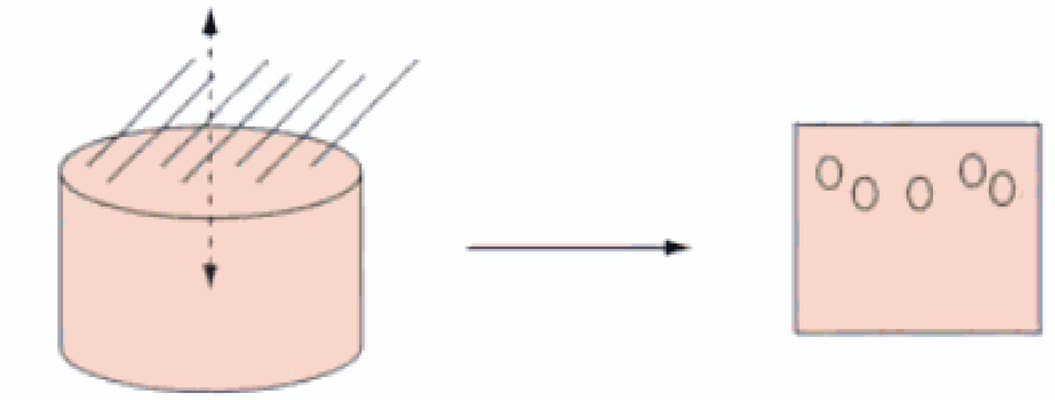
Orientation of skin biopsies



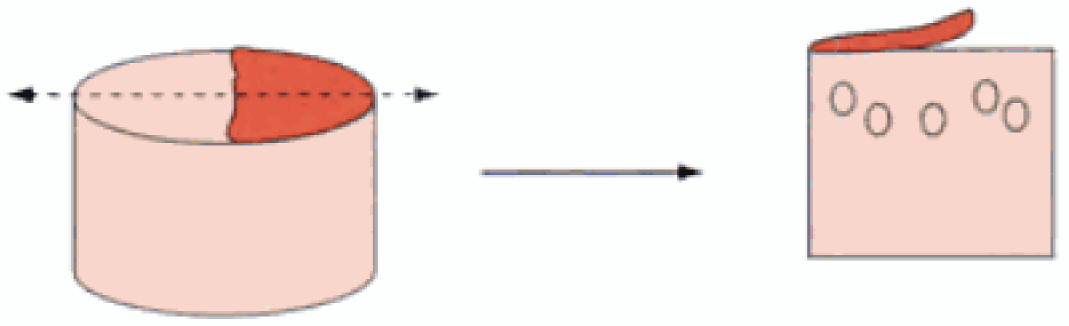
1. Regardless of the care taken to select and obtain skin biopsies, good results will not be obtained if the samples are not correctly oriented. The way a skin biopsy is oriented can mean the difference between a diagnostic and a non-diagnostic sample. Orientation is important because the pathologist has to convert a three dimensional specimen into a microscopic, two dimensional sheet. The pathologist achieves this by first cutting the biopsy specimen in half and then taking thin sections off the flat surface with a microtome. The clinician can ensure that this section is taken in an optimal plane by drawing an arrow (in pencil) on the cardboard on which the biopsy is mounted.



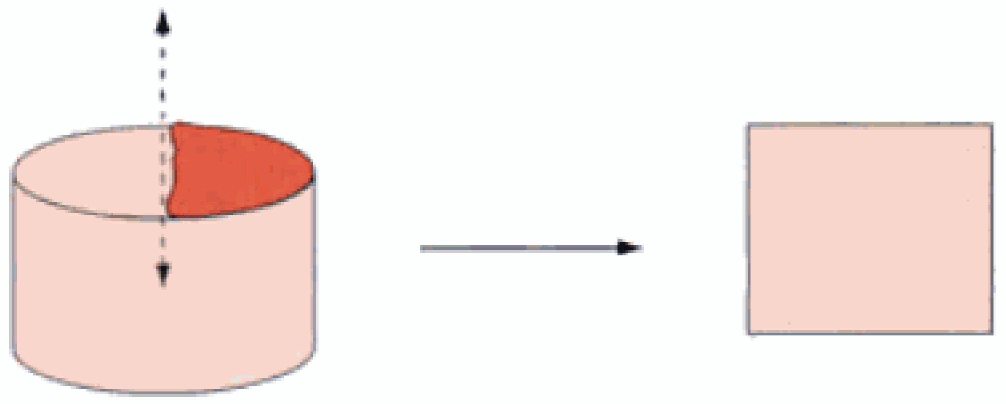
2. In most cases, the correct orientation is provided if the arrow is drawn in the direction of the hair growth. When the pathologist cuts the biopsy in this direction, the resulting section will show the hair follicles in longitudinal section. This allows the pathologist to examine the hair follicles in detail and the epithelium that surrounds them.



3. If the arrow is drawn perpendicular to the direction of hair growth, the resulting section will show the hair follicles in cross section. This prevents an accurate examination of the hair follicles and reduces the amount of epithelium that can be examined. For many diseases, this would prevent a diagnosis from being made. Unfortunately, after the specimen has been fixed, it is virtually impossible for the pathologist to see how the biopsy should be cut. If the clinician hasn't oriented the specimen, the sectioning is left to chance.



4. The only time that specimens are not oriented in the direction of hair growth is when a biopsy has been taken at the junction between normal and affected skin. In this case, the arrow should show the direction that will result in a section showing the transition between the normal and affected areas.



5. If the clinician does not indicate this orientation with an arrow, the pathologist will not be able to determine the correct section to take. After fixation, the biopsy loses its colour and what appeared to have been an obvious junction initially will have completely disappeared. If the cut is then made in the opposite plane, a section of normal skin may be obtained.

Fixation

After collection and orientation, the biopsy should be left sitting on the square of cardboard for approximately 1 minute so that it becomes firmly attached. The biopsy can then be placed in a container of 10% neutral buffered formalin. In the vast majority of cases, the biopsy will remain attached to the cardboard until it reaches the pathologist.

Interpretation of biopsy results

The interpretation of skin biopsies is usually provided in a written report from a pathologist. A histopathology report should contain the following sections:

- A histological description
- A morphological diagnosis
- An aetiological diagnosis if possible
- A comments section

The histological description describes what the pathologist actually sees on the slide. In most cases, the description will describe the stratum corneum and epidermis first, followed by the dermis and the pattern of inflammatory cell infiltrate, and ending with the hair follicles and adnexae (sweat and sebaceous glands). In some cases, if there is a strikingly obvious lesion, such as a tumour, the pathologist will describe this first rather than using the systematic approach described above. The histological description will contain a variety of pathological terms that have great diagnostic significance. However, without many years of experience, the clinician will have to rely on the pathologist to interpret these diagnostic findings.

The morphological diagnosis is a summary of the key pathological changes that have been described in the histological description. This diagnosis will be something similar to "hyperplastic perivascular dermatitis with marked parakeratosis". Again, without dermatopathological training, this part of the report will not be of great meaning to the clinician.

An aetiological diagnosis can be provided if the histological features are characteristic of a particular disease. In the above example, the aetiological diagnosis may state "the changes are consistent with a diagnosis of zinc responsive dermatosis". If this is not possible, the pathologist will provide a list of differential diagnoses in the comments section. This section may also contain other information such as the recommendation to perform further tests such as tissue cultures.

When reading the pathology report, it is important that the clinician correlates the information and the diagnosis with the clinical picture. This is known as clinico-pathological correlation. The clinician should never blindly accept the diagnosis that is written on the report. The pathologist has only seen one tiny section of the animal at one point in time whereas the clinician has access to the whole animal over a series of visits. It is the clinician that makes the diagnosis, not the pathologist. If the clinician feels that the pathological diagnosis does not fit with the clinical picture, the clinician and pathologist should discuss the findings to try and determine an explanation. If there is still a discrepancy, or a diagnosis cannot be made, further biopsies may be indicated to allow examination of additional samples.

Haematology (complete blood count)

A simple diagnostic test in which a sample of whole blood is analysed for abnormalities in the number or appearance of cells.

Technique	Simple
Interpretation	Relatively simple
Specificity	90–100%
Sensitivity	90–100%

Indications

Routine haematology should be performed in dermatological cases in the following circumstances:

- When endocrine diseases are suspected.
- When skin lesions associated with systemic illness are suspected.
- Prior to and during immunosuppressive or cytotoxic therapy.

Technique

A whole blood sample is taken and placed in a tube containing EDTA as an anticoagulant. The sample is analysed either in the practice laboratory or sent to an external laboratory.

Interpretation

Haematological examination provides information about red cells, white cells and platelets. Table 13.3 shows the parameters that usually appear in a haematology profile (CBC). In addition to these values, a haematology report will normally include comments about the appearance of the cells. In some laboratories, abnormal values will be indicated with an asterisk or similar mark. However, clinicians should always examine all the parameters carefully. Clinicians should also be aware that values can be outside the normal range in approximately 5% of healthy animals.

Haematology profiles are interpreted by determining if the values are in the normal range, elevated or decreased. The significance of the various changes can then be assessed.

Table 13.3 Components of a haematology profile

Parameter	Normal Range		Units
	Dog	Cat	
Red cell parameters			
Packed cell volume (PCV)	37–55	24–45	%
Total red blood cell count (RBC)	5.5–8.5	5.0–10.0	$\times 10^{12}$/l
Haemoglobin concentration (Hb)	12–18	8–15	g/dl
Mean corpuscular volume (MCV)	60–77	39–55	fl
Mean corpuscular Hb concentration (MCHC)	32–36	30–36	%
White blood cell parameters			
Total white blood cell count (WBC)	6.0–17.0	5.5–19.5	$\times 10^{9}$/l
Neutrophil count	3.6–12.0	2.5–12.5	$\times 10^{9}$/l
Band neutrophil count	0	0	$\times 10^{9}$/l
Lymphocyte count	0.7–4.8	1.5–7.0	$\times 10^{9}$/l
Eosinophil count	0–1.0	0–1.5	$\times 10^{9}$/l
Monocyte count	0–1.5	0–0.8	$\times 10^{9}$/l
Basophil count	Rare	Rare	$\times 10^{9}$/l
Differential white blood cell count			
Neutrophils	40–80	35–75	%
Band neutrophils	0–3	0–3	%
Lymphocytes	12–30	20–55	%
Eosinophils	2–10	2–12	%
Monocytes	3–10	1–4	%
Basophils	Rare	Rare	%
Platelets			
Platelet count	200–500	300–600	$\times 10^{9}$/l

Haematological changes seen in association with dermatological diseases

Decreased red cell parameters (anaemia)

- Chronic inflammation
- Hypothyroidism
- Systemic lupus erythematosus
- Hyperoestrogenism
- Azathioprine therapy

Increased total WBC count (leukocytosis); Increased neutrophil count (neutrophilia); Increased monocyte count (monocytosis)

- Chronic inflammation
- Stress response
- Hyperadrenocorticism
- Steroid therapy

Increased eosinophil count (eosinophilia); Increased basophil count (basophilia)

- Parasitic infestations
- Allergic inflammation
- Feline eosinophilic granuloma

Increased lymphocyte count (lymphocytosis)

- Chronic infection
- Viral infection
- Immune-mediated disease

Decreased total white blood cell count, neutrophil count and lymphocyte count

- Cytotoxic drug therapy

Decreased eosinophil count (eosinopaenia); Decreased lymphocyte count (lymphopaenia)

- Stress response
- Hyperadrenocorticism
- Steroid therapy

The stress leukogram

The stress leukogram is a collection of haematological changes that are seen in association with stress, hyperadrenocorticism or steroid therapy. A stress leukogram comprises leukocytosis, neutrophilia, eosinopaenia, lymphopaenia and possible monocytosis.

The left shift

A left shift occurs when increased numbers of immature (band) neutrophils are seen in the circulation. A left shift is a characteristic sign of inflammation, especially with infections.

Biochemistry (clinical chemistry)

A simple diagnostic test in which a sample of whole blood is analysed for abnormalities in various biochemical parameters.

Technique	Simple
Interpretation	Relatively simple

Table 13.4 Useful components of a biochemistry profile for dermatological cases

Parameter	Normal Range		Units
	Dog	Cat	
Alanine amino transferase (ALT)	21–102	15–60	IU/L
Alkaline phosphatase (AP) – AMP buffer	20–60	10–100	IU/L
Alkaline phosphatase (AP) – DEA buffer	38–108	18–180	IU/L
Steroid induced isoenzyme of AP – DEA buffer	<35% total	<35% total	IU/L
Bile acids	0–7.0	0–7.0	µmol/l
Total bilirubin	0–6.8	0–6.8	µmol/l
Urea	1.7–7.4	2.8–9.8	mmol/l
Creatinine	40–132	26–118	µmol/l
Glucose	3.0–5.0	3.3–5.0	mmol/l
Cholesterol	3.8–7.0	2.0–3.4	mmol/l
Triglycerides	0.57–1.14	0.57–1.14	mmol/l
Total protein	58–73	69–79	g/l
Albumin	27–37	27–41	g/l
Globulin	18–37	22–56	g/l
Amylase	13–53	28–51	µmol/l
Lipase	13–200	0–83	U/L
Creatinine kinase	50–200	50–200	IU/L
Calcium	2.3–3.0	2.1–2.9	mmol/l
Inorganic phosphate	0.9–1.2	1.4–2.5	mmol/l
Sodium	139–154	145–156	mmol/l
Chloride	99–115	117–140	mmol/l
Potassium	3.6–5.6	4.0–5.0	mmol/l

Guidance Notes

- The normal ranges are only provided for guidance. Clinicians should always use the normal ranges provided by their own laboratory.
- Alkaline phosphatase activity is normally measured in AMP buffer. However, if the steroid induced isoenzyme of alkaline phosphatase is required, the activity is measured in DEA buffer which yields a different normal range.

- Malignant and systemic histiocytosis
- Steroid therapy
- Cytotoxic drug therapy
- Ketoconazole therapy
- Griseofulvin therapy

Decreased ALT is not significant.

AP

AP values are commonly elevated in animals less than 8 months old due to the presence of the bone isoenzyme of alkaline phosphatase. Increased AP in adults is most commonly associated with cholestasis or excessive circulating amounts of glucocorticoids. Increased AP is invariably seen with:

- Hyperadrenocorticism
- Steroid therapy

- Seborrhoea associated with renal failure
- Metastasis of cutaneous tumours to the kidneys (lymphoma)
- Dermatofibrosis associated with renal cystadenocarcinomas
- Renal manifestations of systemic lupus erythematosus
- Aminoglycoside therapy
- Amphotericin B therapy

Decreased BUN may be seen with chronic hepatic insufficiency or severe polydipsia and polyuria. This may be seen with:

- Hepatocutaneous syndrome
- Hyperadrenocorticism

Glucose

The measurement of blood glucose is usually used to detect the presence of diabetes mellitus. Blood glucose can be increased by stress, glucocorticoids or lack of insulin. This may be seen with:

- Diabetes mellitus (may be associated with hepatocutaneous syndrome)
- Hyperadrenocorticism
- Stress (especially cats)
- Acromegaly
- Steroid therapy
- Megestrol acetate therapy

Decreased blood glucose does not suggest any skin diseases.

Cholesterol

The measurement of cholesterol can be used to provide supportive evidence for an endocrinopathy. Increased cholesterol in a fasting sample can be seen with:

- Hypothyroidism (in 70% of cases)
- Hyperadrenocorticism
- Diabetes mellitus
- Steroid therapy

Decreased cholesterol is not significant.

Triglycerides

The measurement of triglycerides can be used to provide supportive evidence for an endocrinopathy. Increased triglycerides in a fasting sample can be seen with:

- Hypothyroidism
- Diabetes mellitus

Decreased triglycerides are not significant.

Total protein, albumin and globulin

Total protein can be decreased by reductions in albumin, globulin or both. Likewise, increased total protein can be caused by elevations in albumin, globulin or both.

Reductions in both albumin and globulin suggest haemorrhage, exudation from severe skin lesions or protein losing enteropathy. This may be seen with:

- Burns
- Toxic epidermal necrolysis
- Erythema multiforme major
- Pemphigus vulgaris
- Food allergy (with concurrent inflammatory bowel disease).

Hypoalbuminaemia alone is usually due to decreased production (liver disease, dietary deficiency or malabsorption), sequestration (body cavity effusions or vasculopathy) or increased loss (protein losing enteropathy or protein losing nephropathy). In dermatological cases, this may be seen with:

- Hepatocutaneous syndrome
- Seborrhoea associated with liver failure
- Malnutrition (which can also lead to seborrhoea)
- Vasculitis
- Food allergy (with concurrent inflammatory bowel disease)
- Glomerulonephritis in association with systemic lupus erythematosus

Hypoglobulinaemia can be caused by liver disease, nephrotic syndrome, immunodeficiencies, or immunosuppression. This may be seen with:

- Hepatocutaneous syndrome
- Seborrhoea associated with liver failure
- Glomerulonephritis in association with systemic lupus erythematosus
- Primary immunoglobulin deficiencies
- Immunosuppressive therapy

Hyperalbuminaemia is usually due to dehydration. This would be a rare finding in dermatological cases.
Hyperglobulinaemia may be associated with parasitism, chronic inflammation, immune-mediated diseases or neoplasia. This may be seen with:

- Severe flea allergy
- Scabies
- Demodicosis
- Deep pyoderma
- Other cutaneous infections (nocardiosis, actinomycosis, actinobacillosis)
- Deep fungal infections
- Leishmaniasis
- Pemphigus
- Lupus
- Myeloma

Amylase and lipase

The measurement of amylase and lipase is used to assess diseases of the pancreas. Increases in amylase and lipase suggest pancreatitis. Amylase and lipase are rarely elevated in dermatological diseases but could possibly be seen with:

- Feline paraneoplastic alopecia (caused by pancreatic adenocarcinoma)
- Panniculitis (may be caused by pancreatic damage)
- Hepatocutaneous syndrome (may be caused by a pancreatic tumour)

Decreases in amylase and lipase are not significant.

Creatine kinase (CK)

The measurement of creatine kinase is used as a crude indicator of muscle damage, although it has poor sensitivity and specificity for spontaneous muscle disease. In dermatological cases, CK may be elevated in:

- Dermatomyositis
- Systemic lupus erythematosus
- Hypothyroidism
- Hyperadrenocorticism

Decreased CK is not significant.

Calcium and inorganic phosphate

Many conditions can lead to alterations in blood calcium and phosphate concentrations. Primary hyperparathyroidism and certain malignancies (lymphosarcoma, anal sac adenocarcinomas) lead to elevated calcium and decreased phosphate. Renal failure, Vitamin D toxicity, and Addison's disease can lead to elevations in both calcium and phosphate. As most of these conditions do not have cutaneous manifestations, a complete discussion of the causes of hyper- and hypo-calcaemia and phosphataemia is beyond the scope of this text. However, in dermatological cases, hypercalcaemia may be seen with:

- Lymphosarcoma (due to secretion of a parathyroid hormone-like substance)
- Anal sac adenocarcinoma (due to secretion of a parathyroid hormone-like substance)
- Hypoadrenocorticism (dogs undergoing treatment for hyperadrenocorticism)
- Renal failure (which may lead to seborrhoea)
- Blastomycosis (or other granulomatous diseases)

Hypocalcaemia is not associated with dermatological cases.

Sodium, chloride and potassium

Abnormalities of electrolyte concentrations can be seen with many diseases, but rarely in dermatological cases. Hence, electrolyte disturbances will not be discussed in detail here. However, when treating skin conditions, clinicians should always be aware of the possibility of inducing a state of hypoadrenocorticism. This may occur with:

- Treatment of hyperadrenocorticism with Mitotane
- Treatment of hyperadrenocorticism by adrenalectomy
- Abrupt cessation of steroid therapy after prolonged use

Hypoadrenocorticism leads to decreased sodium (hyponatraemia), decreased chloride (hypochloraemia) and increased potassium (hyperkalaemia). This leads to a reduced sodium:potassium ratio of less than 25:1.

Tests to assess thyroid function

The assessment of thyroid function is based on the measurement of various thyroid gland related hormones. These include thyroxine (T4), tri-iodothyronine (T3), free T4, and thyroid stimulating hormone (TSH). These hormones are normally measured as basal concentrations in the serum but T4 can be measured before and after stimulation of the thyroid gland. In some circumstances, the measurement of autoantibodies directed against T3 and T4 is also indicated.

Measurement of basal total T4 and T3 concentrations

Assessment of total T4 concentrations is the simplest and most economical way to evaluate thyroid gland function. Although endocrine laboratories can also measure total T3 concentrations, there is little benefit in performing this test in addition to the T4 measurement.

Technique	Simple
Interpretation	Requires caution and experience
Specificity	50–80%
Sensitivity	90%

Indications

Indicated in dogs presenting with the following:

- Clinical signs consistent with hypothyroidism
- Persistent, non-pruritic, symmetrical alopecia
- Unexplained dull coat
- Persistent seborrhoea
- Recurrent pyoderma
- Adult onset demodicosis
- Persistent, unexplained lethargy

Indicated in cats presenting with signs consistent with hyperthyroidism.

Technique

A blood sample is taken and either serum or plasma is submitted to an endocrine laboratory.

Interpretation

The normal ranges of total T4 and T3 are shown in Table 13.5.

These ranges are only provided as a guide and clinicians should always consult the values provided by their own laboratory. Sight hounds and hunting hounds (Saluki, Deerhound, Wolfhound and Afghan) have lower T4 and higher TSH concentrations than other breeds.

Table 13.5 Normal ranges for parameters measured in thyroid function tests

Parameter	Normal Range		Units
	Dog	Cat	
Thyroxine (T4)	13–52	13–48	nmol/l
Tri-iodothyronine (T3)	0.3–2.5	0.9–3.1	nmol/l
Free T4	7–30	5–25	pmol/l
Thyroid stimulating hormone (TSH)	< 0.4	N/A	ng/ml

Clinicians should take great care when interpreting T4 and T3 values that fall outside these ranges. Many non-thyroidal illnesses and drugs can suppress thyroid hormone concentrations. Hence a low T4 or T3 concentration IS NOT specific for hypothyroidism. The presence of low thyroid hormone concentrations in association with other diseases is referred to as the euthyroid sick syndrome. This common phenomenon is the major reason for overdiagnosis of hypothyroidism and administration of inappropriate treatment. Conditions known to cause the euthyroid sick syndrome include:

- Pneumonia
- Immune-mediated haemolytic anaemia
- Systemic lupus erythematosus
- Intervertebral disc disease
- Generalised demodicosis
- Deep pyoderma
- Deep fungal infections
- Hyperadrenocorticism
- Diabetes mellitus
- Renal failure
- Liver failure
- Cardiac failure
- Gastrointestinal diseases
- Lymphosarcoma

Drugs known to decrease thyroid hormone concentrations include:

- Glucocorticoids
- Potentiated sulphonamides
- Diazepam
- Androgens
- Aspirin
- Phenylbutazone
- Phenobarbital
- Primidone

The significance of a low T4 concentration depends on the actual value and the presence or absence of other supportive evidence. The examples shown in Table 13.6 will help to illustrate this principle. Note that as more evidence is gathered, the likelihood of the test diagnosing hypothyroidism increases (i.e. the specificity increases). This principle should still be followed if very low total T4 concentrations are obtained because these may result from severe euthyroid sick syndrome. If the clinician is unable to obtain sufficient evidence to make a definitive diagnosis of hypothyroidism based on the measurement of total T4 and other clinical information, additional tests should be performed. These may include measurement of TSH concentrations or the TSH stimulation test.

Clinicians should also be careful when normal T4 values are obtained in suspected cases of hypothyroidism. This can occur in the early stages of the disease when thyroxine concentrations are fluctuating into and out of the normal range. In this case, further function tests are indicated.

Measurement of free T4 concentrations

Free T4 is the metabolically active fraction of T4. As free T4 is non-protein bound, it is less affected by diseases or drugs that affect plasma proteins. However, despite this theoretical advantage, there does not currently appear to be a clear diagnostic benefit in measuring free T4 as compared to total T4.

Technique	Simple
Interpretation	Requires caution and experience
Specificity	50–80%
Sensitivity	90%

Table 13.6 **Guidelines for interpreting basal total T4 concentrations**

Clinical information known about a case	Interpretation
T4 = 10 nmol/l	Could be: ● A normal dog ● A dog with euthyroid sick syndrome ● A dog receiving the drugs listed above ● A dog with hypothyroidism
T4 = 10 nmol/l and cholesterol is increased	More likely to be hypothyroidism, but could still be: ● A dog with euthyroid sick syndrome ● A dog receiving the drugs listed above
T4 = 10 nmol/l and cholesterol is increased and dog is slightly anaemic	Even more likely to be hypothyroidism, but could still be: ● A dog with euthyroid sick syndrome ● A dog receiving the drugs listed above
T4 = 10 nmol/l and cholesterol is increased and dog is slightly anaemic and dog has clinical signs consistent with hypothyroidism	Highly likely to be hypothyroidism, but could still be: ● A dog with euthyroid sick syndrome ● A dog receiving potentiated sulphonamides
T4 = 10 nmol/l and cholesterol is increased and dog is slightly anaemic and dog has clinical signs consistent with hypothyroidism and dog responds completely to thyroid supplementation	Dog has hypothyroidism

Indications

Can be used as an alternative to the measurement of total T4. Indicated in dogs with:

- Clinical signs consistent with hypothyroidism
- Persistent, non-pruritic, symmetrical alopecia
- Unexplained dull coat
- Persistent seborrhoea
- Recurrent pyoderma
- Adult onset demodicosis
- Persistent, unexplained lethargy

Technique

A blood sample is taken and either serum or plasma is submitted to an endocrine laboratory.

Interpretation

The normal range for free T4 is shown in Table 13.5. The principles underlying the interpretation of free T4 concentrations are the same as those for total T4. A low free T4 concentration IS NOT diagnostic for hypothyroidism and clinicians should follow the guidelines in Table 13.6 before making a definitive diagnosis.

Measurement of TSH concentrations

Assays to measure circulating canine TSH are now validated and performed at commercial endocrine laboratories.

Technique	Simple
Interpretation	Requires caution
Specificity	80%
Sensitivity	90%

Indications

Circulating TSH is often measured at the same time as total T4 in order to provide further evidence for hypothyroidism. Hence, TSH can be measured in dogs with the following:

- Clinical signs consistent with hypothyroidism
- Persistent, non-pruritic, symmetrical alopecia
- Unexplained dull coat
- Persistent seborrhoea
- Recurrent pyoderma
- Adult onset demodicosis
- Persistent, unexplained lethargy
- Previously recorded low T4 concentrations

Technique

A blood sample is taken and either serum or plasma is submitted to an endocrine laboratory.

Interpretation

The normal range for TSH is shown in Table 13.5. In primary hypothyroidism, TSH concentrations are usually elevated to above 1.0 ng/ml because the pituitary gland is trying to increase thyroid hormone production from the failing thyroid glands. Hence a high TSH concentration in association with a low T4 or free T4 concentration is suggestive of hypothyroidism. However, clinicians should be aware that this combination of changes can still be seen with euthyroid sick syndrome. Therefore, the diagnosis of hypothyroidism should still be based on the principles outlined in Table 13.6, with the TSH concentration being regarded as an additional piece of evidence.

The TSH stimulation test

The TSH stimulation test is currently the most accurate method for assessing canine thyroid gland function.

Technique	Simple, as long as TSH is available
Interpretation	Requires caution and experience
Specificity	95% (currently the most specific test for hypothyroidism, but abnormal results may rarely be found in severe non-thyroidal illness or following treatment with potentiated sulphonamides)

Sensitivity	98% (currently the most sensitive test for hypothyroidism, but may be normal in very early cases)

Indications

The TSH stimulation test can be used as a primary screening test for hypothyroidism. When used in this way, the test is indicated in dogs presenting with the following:

- Clinical signs consistent with hypothyroidism
- Persistent, non-pruritic, symmetrical alopecia
- Unexplained dull coat
- Persistent seborrhoea
- Recurrent pyoderma
- Adult onset demodicosis
- Persistent lethargy.

However, TSH is not routinely available and it can be difficult locating a commercially available source for diagnostic testing. Some endocrine laboratories have access to TSH and will supply it to clinicians wishing to perform this test. Alternatively, many University hospitals and speciality practices will perform TSH stimulation tests on referred cases.

In view of these difficulties, the TSH stimulation test is probably best reserved for cases in which a diagnosis cannot be made on the basis of history, clinical signs, haematological and biochemical changes, and total T4 and TSH concentrations (see Table 13.6). It should be noted that if these principles are applied, the use of the TSH stimulation test is rarely required.

Technique

1. Collect a blood sample into a plain or heparinised tube.
2. Inject 0.1 IU/kg bovine TSH intravenously.
3. Collect a second blood sample 6 hours later.
4. The total T4 concentration is measured in both samples.
5. If a TSH stimulation test is required in a dog that is already receiving thyroid hormone supplementation (to investigate a suspected incorrect diagnosis), the drug should be discontinued for 8 weeks to allow the suppressed thyroid glands to recover.

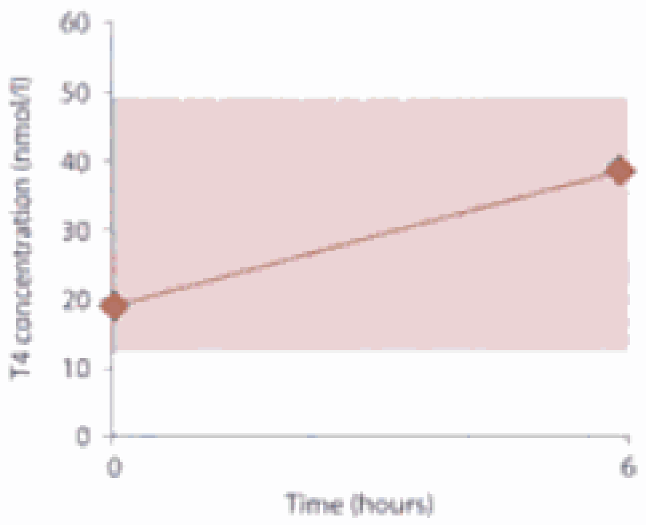
Interpretation

Although the TSH stimulation test is currently the most reliable method for assessing thyroid gland function, the interpretation of the results requires some caution. Most endocrine laboratories will provide their own guidelines for interpreting the test, but the following guidelines should be useful to clinicians when analysing these results.

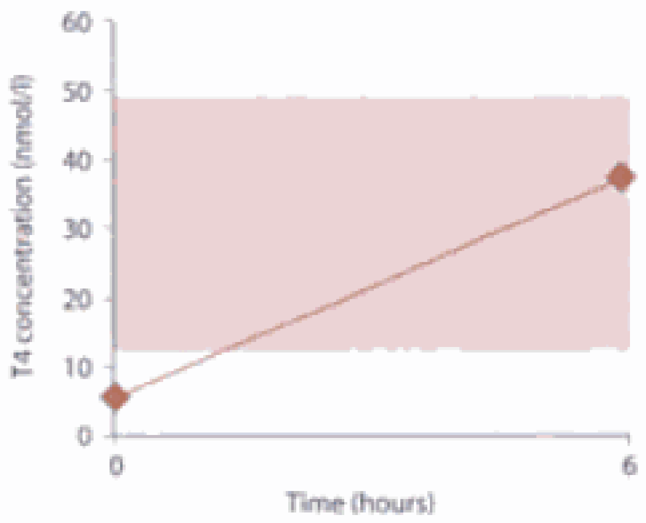
The results of the TSH stimulation test are interpreted by comparing the T4 concentration in the second sample with that in the first. A normal thyroid gland will respond to the TSH by secreting T4 during the 6 hour period between the two blood samples. With normal thyroid function, the concentration in the second sample should be at least 1.5 times that of the first sample and be above 35 nmol/l. However, some care is warranted when applying this rule. The following graphs illustrate various results that can be obtained with the TSH stimulation test, and their interpretation. The shaded areas represent the normal range for T4 concentrations in the dog.

Results

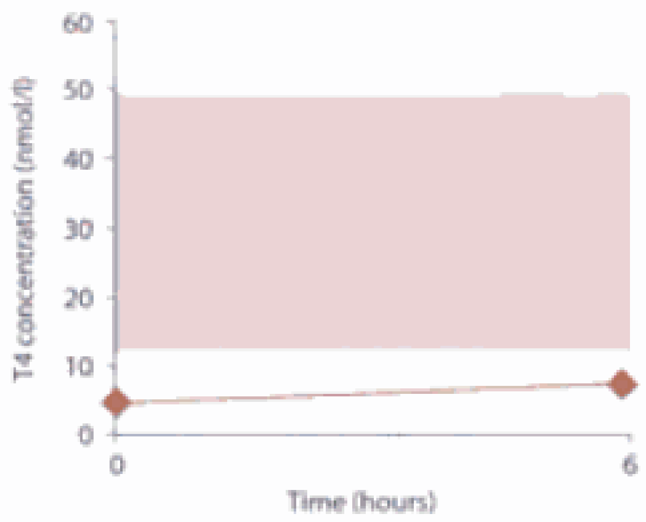
Interpretation



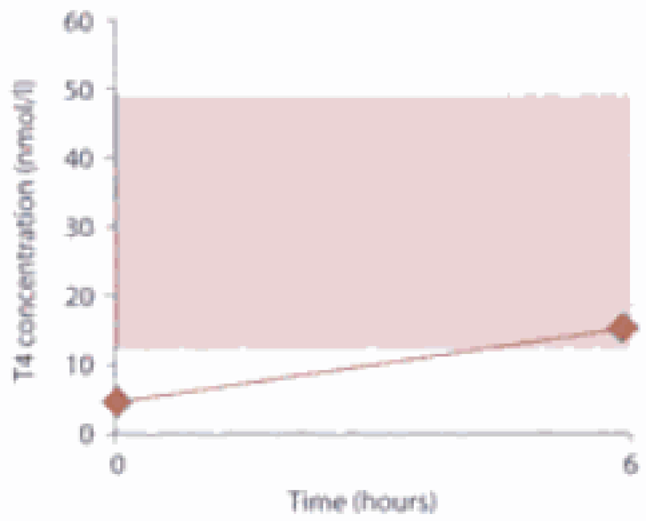
The T4 concentration has increased from 20 nmol/l to 40 nmol/l. This indicates normal thyroid gland function.



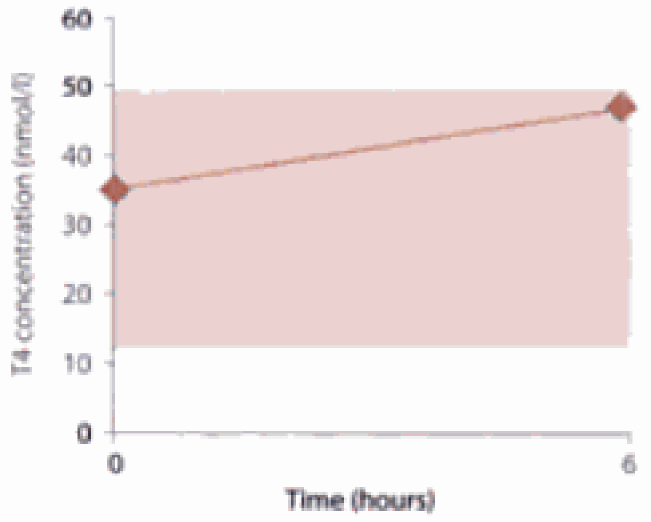
The T4 concentration has increased from 7 nmol/l to 38 nmol/l. This indicates normal thyroid gland function. In this case, the initially low T4 concentration must have been due to non-thyroidal factors (other illnesses, drugs). Thyroid supplementation is not indicated.



The T4 concentration has increased from 5 nmol/l to 10 nmol/l. Although the T4 concentration has doubled, it hasn't reached 35 nmol/l. In conjunction with appropriate clinical signs, this is diagnostic for hypothyroidism and thyroid supplementation is indicated.



The T4 concentration has increased from 5 nmol/l to 22 nmol/l. In this case the T4 concentration has increased more than 4 times but it hasn't reached 35 nmol/l. Although this result would normally indicate hypothyroidism, the diagnosis should be substantiated by further clinical and biochemical evidence. If therapy is initiated, clinicians should monitor for a rapid clinical response.



In this case, the T4 concentration has increased from 36 nmol/l to 48 nmol/l. According to the normal criteria for assessing the test, this dog would be hypothyroid because the second value isn't 1.5 times greater than the first. However, this dog is very unlikely to be hypothyroid because both values are well in the normal range. If the clinical signs are strongly suggestive of hypothyroidism, a blood sample should be submitted for measurement of T4 autoantibodies (see below).

The TRH stimulation test

The TRH stimulation test works by stimulating TSH release from the pituitary gland. This then stimulates the release of T4 from the thyroid glands which can be measured. However, the increases seen with the TRH stimulation test are less than those seen with the TSH stimulation test and it is therefore less reliable.

Technique	Simple
Interpretation	Requires caution and experience
Specificity	50–75% (abnormal results may be found in normal dogs and dogs with other diseases)
Sensitivity	95% (hypothyroid dogs usually fail to respond to TRH but a normal result may be found in early cases)

Indications

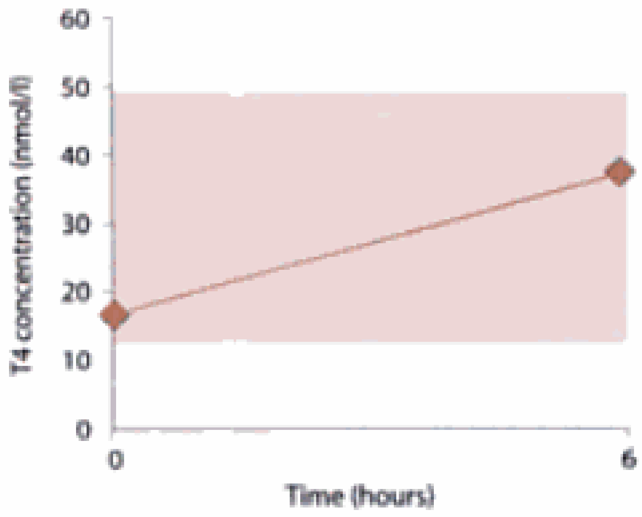
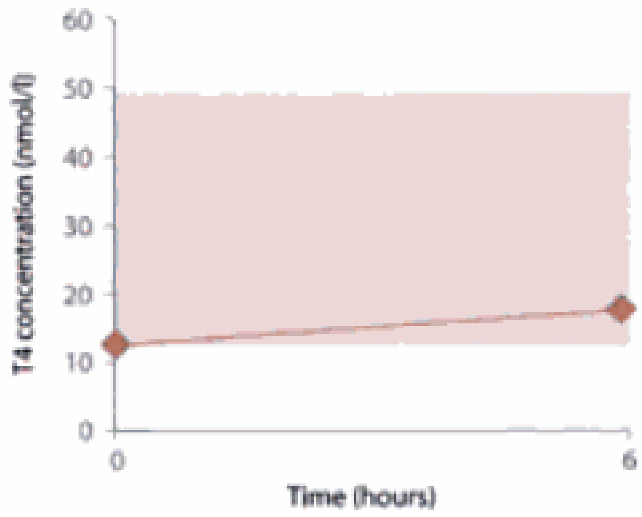
The indications for the TRH stimulation test are the same as for the TSH stimulation test. It can also be used in situations where a stimulation test is required but TSH is not available (TRH is usually easier to acquire than TSH).

Technique

1. Collect a blood sample into a plain or heparinised tube.
2. Inject 0.02 mg/kg bovine TRH intravenously slowly over 1 minute.
3. Collect a second blood sample 4 hours later.
4. The total T4 concentration is measured in both samples.

Interpretation

The results of the TRH stimulation test are interpreted by comparing the T4 concentration in the second sample with that in the first. A normal thyroid gland will respond to the TSH release stimulated by the TRH by secreting T4 during the 4 hour period between the two blood samples. With normal thyroid function, the concentration in the second sample should be at least 1.2 times that of the first sample and be above 30 nmol/l. Similar care should be taken in interpreting the results of TRH stimulation tests as described for TSH stimulation tests. However, in addition, clinicians should be aware of the lack of specificity of this test compared to the TSH stimulation test. The following graphs illustrate this problem. The shaded areas represent the normal range for T4 concentrations in the dog.

Result	Interpretation
	The T4 concentration has increased from 20 nmol/l to 40 nmol/l. This indicates normal thyroid gland function. A normal TRH stimulation test is therefore useful for ruling out hypothyroidism.
	The T4 concentration has increased from 10 nmol/l to 14 nmol/l. This result might indicate hypothyroidism but it could also be seen in normal dogs, dogs with other illnesses or dogs receiving various drugs. Further historical, clinical, haematological and biochemical evidence is required before a diagnosis of hypothyroidism can be made. An abnormal TRH stimulation test is therefore not specific for hypothyroidism.

Measurement of T3 and T4 autoantibodies

Dogs with hypothyroidism will occasionally develop autoantibodies against T3 or T4. These antibodies can interfere with the assays that are used to measure thyroid hormones, resulting in incorrectly high results.

Technique	Simple
Interpretation	Simple
Specificity	98%
Sensitivity	98%

Indications

T3 and T4 autoantibodies should be measured in cases that fulfil the following criteria:

- The dog shows historical, clinical, haematological and biochemical evidence of hypothyroidism
- Tests performed to measure T3 and T4 concentrations have yielded unexpectedly high results

Technique

A blood sample is taken and serum is submitted to an endocrine laboratory.

Interpretation

The laboratory will report if T3 and T4 autoantibodies are present or not. If they are, their presence may explain the previously obtained, spuriously high results. However, their presence alone does not confirm a diagnosis of hypothyroidism. In cases with T3 and T4 autoantibodies, the clinician must weigh up the available evidence and decide if thyroid hyperadrenocorticism and decreased in hypoadrenocorticism. Hypoadrenocorticism may be seen with excessive administration of glucocorticoids, Addison's disease or following treatment of hyperadrenocorticism. Note that in some texts, the term "iatrogenic hyperadrenocorticism" is used to describe the syndrome produced by excessive glucocorticoid therapy. Although the clinical signs of this syndrome are identical to naturally occurring hyperadrenocorticism, the adrenal glands are atrophic and not hyper-secretory so the use of that terminology is not appropriate.

Technique	Simple
Interpretation	Requires caution and experience
Specificity	90%
Sensitivity	85% (pituitary dependant), 55% (adrenal dependant), 95% (iatrogenic)

Indications

The ACTH stimulation test is indicated in dermatological cases in the following circumstances:

- As a diagnostic test in dogs with clinical signs consistent with hyperadrenocorticism
- In dogs with clinical signs consistent with excessive glucocorticoid therapy
- To monitor dogs on treatment for hyperadrenocorticism
- To monitor dogs after adrenalectomy
- As an additional test if a low dose dexamethasone suppression test was inconclusive.

Technique

1. Collect a blood sample into a heparinised tube.
2. There are two main methods for stimulation.
   i) Inject 0.25 mg synthetic ACTH (Synacthen, Ciba Laboratories) intravenously (regardless of body weight). Alternatively, some authors have recommended a dose of 0.1 mg ACTH for dogs less than 10 kg and 0.2 mg for dogs greater than 10 kg.
   ii) OR Inject 2.2 IU/kg ACTH gel intramuscularly.
3. Collect a second blood sample 2 hours later.
4. The cortisol concentration is measured in both samples.
5. If an ACTH stimulation test is required in a dog that is already receiving prednisolone, the drug should be discontinued for 48 hours if possible so that it does not cross react with the assay (care should be taken not to induce an Addisonian crisis).

Interpretation

The results of the ACTH stimulation test are interpreted by comparing the cortisol concentration in the second sample with that in the first. A normal adrenal gland will respond to the ACTH by secreting cortisol during the 2 hour period between the two blood samples. This usually results in a 2–3 fold increase in cortisol concentrations. However, in hyperadrenocorticism, this response should be exaggerated. In dogs receiving excessive glucocorticoids, the concentration in the second sample is usually the same as that of the first sample due to the adrenal gland suppression.

The ranges for cortisol concentrations before and after ACTH stimulation are shown for various conditions in Table 13.7. As with other sections in this chapter, these ranges are only provided as a guide and clinicians should always consult the values provided by their own laboratory.

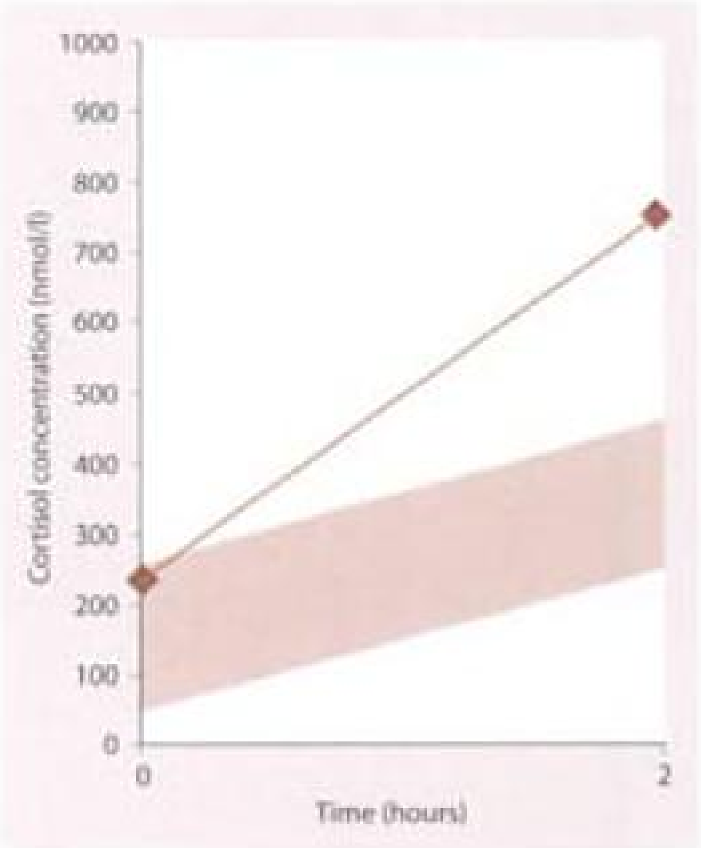
Table 13.7 Reference ranges for cortisol concentrations in the dog

Condition	Cortisol concentration		Units
	Resting	Post ACTH	
Normal dog	33–281	136–415	nmol/l
Excessive glucocorticoid treatment	< 10–50	< 10–135	nmol/l
Borderline hyperadrenocorticism	33 – >281	415–600	nmol/l
Hyperadrenocorticism	33 – >281	> 600	nmol/l

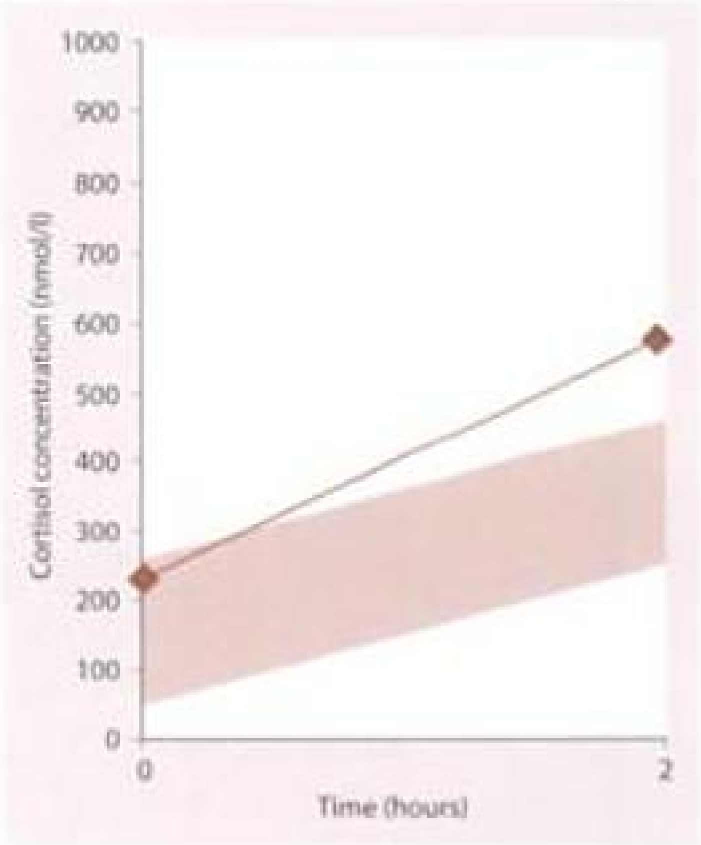
The following graphs illustrate some results that could be obtained with the ACTH stimulation test. The shaded areas represent the normal range for pre- and post-ACTH cortisol concentrations in the dog.

Result

Interpretation



In this case, the cortisol concentration has increased from the upper end of the normal range to over 700 nmol/l. In conjunction with consistent clinical signs, this would be diagnostic for hyperadrenocorticism. However, this test cannot distinguish between pituitary and adrenal dependant hyperadrenocorticism.



In this case, the cortisol concentration has increased from the upper end of the normal range to just over 500 nmol/l. This could be consistent with hyperadrenocorticism but could also be seen in stressed dogs or dogs with other diseases.

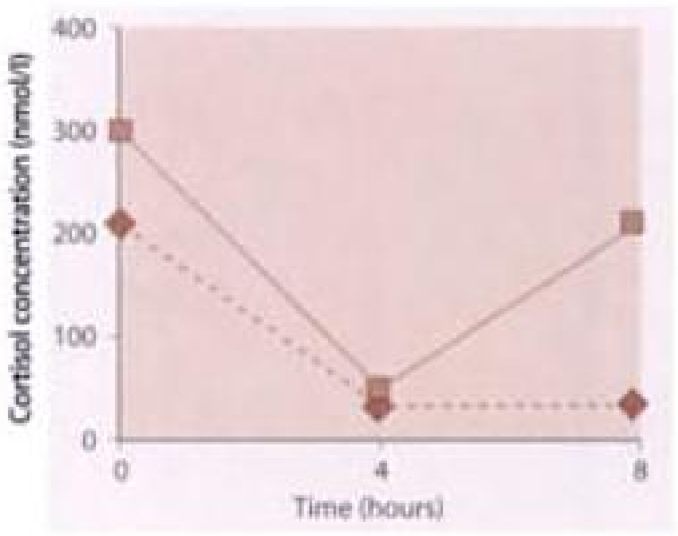
2. Inject 0.015 mg/kg dexamethasone intravenously.
3. Collect additional blood samples at 4 and 8 hours.
4. The cortisol concentration is measured in all three samples.

Interpretation

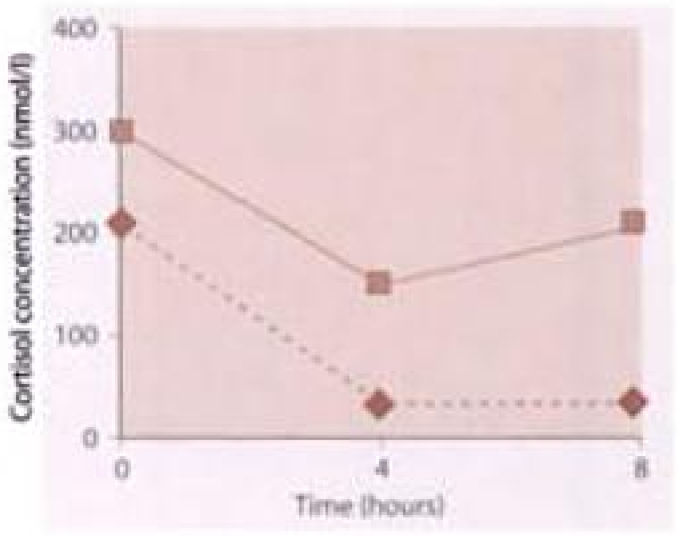
The test is interpreted by comparing the cortisol concentrations in the 4 and 8 hour samples with the basal concentration. In normal dogs, the cortisol concentration is suppressed at both 4 and 8 hours to less than 40 nmol/l. In hyperadrenocorticism, the cortisol concentration will fail to be suppressed at 4 hours, 8 hours or both. Five patterns of suppression have been described in dogs with hyperadrenocorticism (Peterson ME, Vet Clin N Am, 14:739, 1984). These patterns are described in the following graphs.

Result

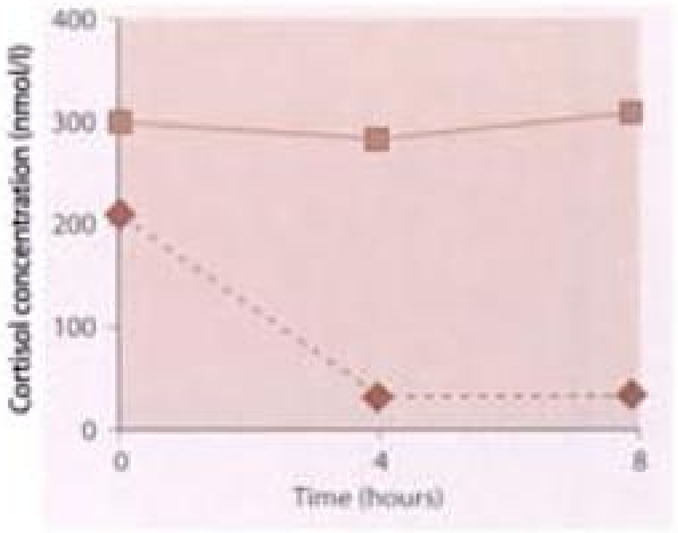
Interpretation



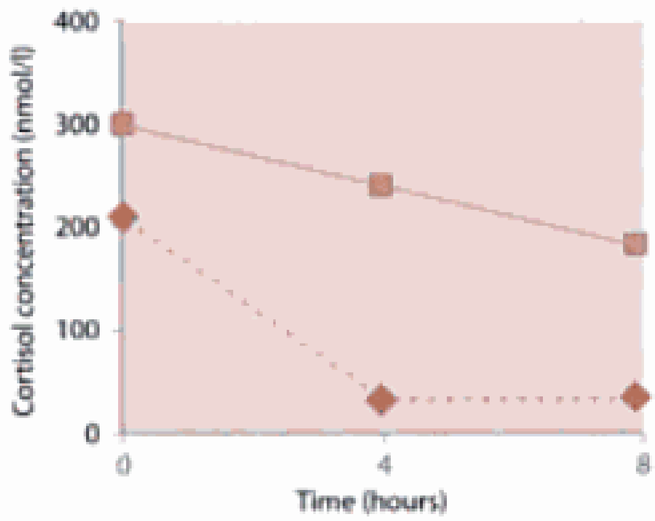
The *dotted* line represents the response seen in normal dogs. The *solid* line represents the response seen in a dog with hyperadrenocorticism. The cortisol concentration has been suppressed to less than 40 nmol/l at 4 hours but it has increased again by 8 hours. This pattern is seen in 30% of dogs with pituitary dependant hyperadrenocorticism but 0% of dogs with adrenal tumours. Hence, if this pattern is seen, a definitive diagnosis can be made and no further differentiating tests are required.



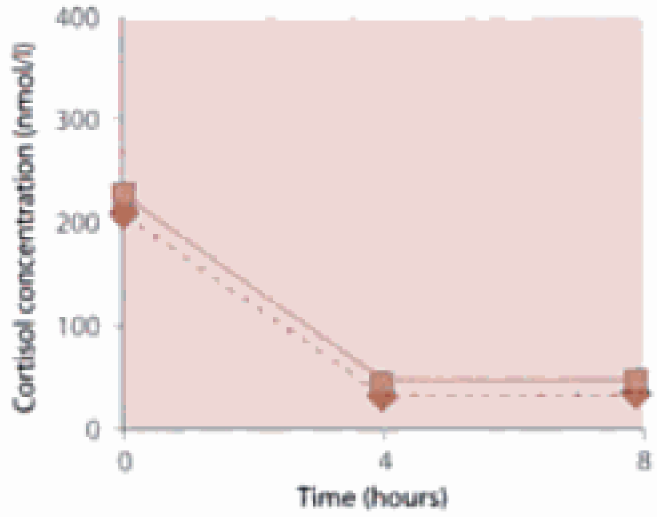
The cortisol concentration has been suppressed by about 50% at 4 hours but not to less than 40 nmol/l. At 8 hours, the concentration has nearly returned to resting values. This pattern is seen in 25% of dogs with pituitary dependant hyperadrenocorticism and 5% of dogs with adrenal tumours. A further differentiating test is therefore required. *Dotted* line represents response seen in normal dogs.



In this case, the cortisol concentration has not been suppressed at either 4 or 8 hours. This pattern is seen in 25% of dogs with pituitary dependant hyperadrenocorticism and 80% of dogs with adrenal tumours. A further differentiating test is required. *Dotted* line represents response seen in normal dogs.



The cortisol concentration has been suppressed by about 50% at 8 hours but it is still above 40 nmol/l. This pattern in seen in 15% of dogs with pituitary dependant hyperadrenocorticism and 15% of dogs with adrenal tumours. A further differentiating test is required. *Dotted* line represents response seen in normal dogs.



In this case, the cortisol concentration has been suppressed to less than 40 nmol/l at both 4 and 8 hours. This is the response generally seen in normal dogs, but it can be seen in 5% of dogs with pituitary dependant hyperadrenocorticism. If hyperadrenocorticism is strongly suspected, the clinician can either:

- Perform an ACTH stimulation test
- Wait for 2 - 4 months and repeat the test.

The ACTH stimulation test versus the low dose dexamethasone suppression test – which is best?

Clinicians and students often enquire which of these two tests is the best. The answer is that neither test is completely superior to the other. The ACTH stimulation test is quicker to perform and is slightly more specific than the low dose dexamethasone suppression test. It is also the test of choice for iatrogenic hyperglucocorticoidism. A further advantage is that it provides a baseline for subsequent monitoring of treatment. However, the low dose dexamethasone suppression test is more sensitive and is less likely to miss the diagnosis. There is also a 30% chance that the low dose dexamethasone suppression test will distinguish between pituitary and adrenal forms of hyperadrenocorticism (see the first graph above). Hence, both tests have their advantages and clinicians must decide for themselves which test to choose first. Regardless of this choice, some cases will require both tests in order to confirm the diagnosis.

The high dose dexamethasone suppression test

The high dose dexamethasone suppression test is based on the same principles as the low dose dexamethasone suppression test. However, by using a higher dose of dexamethasone, it is possible to suppress cortisol secretion in the majority of cases of pituitary dependant hyperadrenocorticism. This does not occur with adrenal tumours. Hence, the high dose dexamethasone suppression test is used to differentiate between the two causes.

Technique	Simple
Interpretation	Requires caution and experience
Specificity	80–90%
Sensitivity	80–90%

Techniques	Require considerable expertise and equipment
Interpretation	Requires extensive expertise and experience
Specificity	Varies depending on the modality
Sensitivity	Varies depending on the modality

Indications

The use of diagnostic imaging techniques in the investigation of hyperadrenocorticism varies between institutions. Although widely available, abdominal radiography is not very sensitive at detecting adrenal masses. It should therefore be reserved for cases in which diagnosis is proving difficult. Abdominal ultrasonography is very useful for detecting unilateral or bilateral enlargement of the adrenal glands, but it requires a skilled ultrasonographer. However, it can play an important role in the differentiation of the two causes, especially if diagnosis is proving a problem. The access to CT and MRI scans is limited at present although they are becoming more widely available. These modalities can provide images of both the adrenals and the pituitary gland. Imaging the pituitary gland is most appropriate in dogs that are also displaying neurological signs. These techniques may play an increasingly important role in the investigation of hyperadrenocorticism in the future.

Techniques

Clinicians should consult appropriate imaging texts for further information on these techniques.

Interpretation

The interpretation of adrenal and pituitary images with any of the modalities described above should be performed by a qualified and skilled diagnostic imager.

Miscellaneous endocrine tests

Sex hormone stimulation test

The sex hormone stimulation test involves the measurement of adrenal sex steroids before and after the administration of ACTH.

Technique	Simple
Interpretation	Requires caution and experience
Specificity	Not known
Sensitivity	Not known

Indications

The sex hormone stimulation test is indicated in dogs with suspected adrenal sex hormone alopecia (alopecia X, growth hormone responsive dermatosis).

Technique

1. Collect a blood sample into a heparinised tube.
2. There are two main methods for stimulation.
   i) Inject 0.25 mg synthetic ACTH (Synacthen, Ciba Laboratories) intravenously (regardless of body weight). Alternatively, some authors have recommended a dose of 0.1 mg ACTH for dogs less than 10 kg and 0.2 mg for dogs greater than 10 kg.
   ii) OR Inject 2.2 IU/kg ACTH gel intramuscularly.

TREATMENT OF SUPERFICIAL ECTOPARASITES

Treatment objectives

1. To eliminate the parasitic infestation.
2. To provide short-term symptomatic treatment if the pruritus is severe.

Treatment principles

1. Treat the affected animal with an appropriate anti-parasitic preparation (Table 14.1). The duration of treatment should be longer than the length of the parasite's life cycle to ensure complete eradication.
2. If the parasite is known to be contagious (lice, *Sarcoptes*, *Cheyletiella*, *Notoedres*), treat in-contact animals with the same product. This includes other dogs and cats in the house as well as those that the animal visits. This reduces or eliminates a source of cross infestation.

Table 14.1 Products currently recommended for the treatment of superficial ectoparasites. The numbers indicate the recommended order in which the products should be used.

Parasite	Product
Sarcoptes scabiei	1. Selamectin 2. Amitraz Lime sulfur 3. Fipronil 4. Ivermectin Milbemycin
Cheyletiella yasguri	1. Pyrethroid shampoo Selenium sulphide Fipronil Selamectin Lime sulfur 2. Amitraz (dogs only) 3. Ivermectin
Notoedres cati	1. Selamectin Lime sulfur 2. Fipronil 3. Ivermectin
Trombicula autumnalis	1. Fipronil
Lice	1. Fipronil 2. Imidacloprid
Ticks	1. Fipronil Amitraz collar

3. If appropriate, treat the animal's environment with an insecticidal spray to kill any parasites that have temporarily fallen off the host (lice, *Sarcoptes*, *Cheyletiella*, *Notoedres*).
4. If necessary, the pruritus can be treated symptomatically with a 5–7 day course of glucocorticoids. Prednisolone can be administered at a dose of 1 mg/kg daily for 3–5 days followed by 2 days at 0.5 mg/kg. This is only likely to be necessary when treating scabies.
5. Reinspect the animal after 3–4 weeks to confirm response or resolution.

Products for the treatment of superficial ectoparasites

The products that can be used to treat superficial ectoparasites are listed in Table 14.1 and described below. The numbers in the table provide a guide as to the recommended order in which the products should be used. These recommendations are based on such factors as licensed indications, known efficacy, safety and ease of use. The sequential use of products is indicated in situations where the first product has proved to be ineffective.

Clinicians should be aware that the unlicensed use of products such as ivermectin and milbemycin is illegal in some countries. In the UK and USA, these products should only be used according to the following guidelines:

- Clinicians should first use a product specifically licensed (labelled) for the condition.
- If no product is specifically licensed for the condition, clinicians should try a product licensed for that species for a similar condition.
- If both of the above products are clinically ineffective, the extra label use of an unlicensed product is appropriate as long as there is a valid client/patient/veterinarian relationship.
- Clinicians should obtain written client consent when using products in an extra-label manner.

Clinicians should also be aware of the requirements for the safe use and disposal of insecticidal products. Veterinary staff and owners must always read the product labels carefully to ensure that these chemicals are used safely and disposed of in a way that will not contaminate the environment or waterways.

Selamectin

Trade names: Stronghold® (UK), Revolution® (USA).
Formulation: Spot-On for topical use.

Dose and route of administration:

1. For the treatment of scabies, cheyletiellosis and notoedric mange, two doses of selamectin should be administered a month apart.
2. Selamectin is applied by emptying the contents of an appropriate sized tube onto the skin on the back of the neck in front of the shoulder blades. Animals can be bathed 2 hours after application of selamectin without loss of efficacy.
3. Selamectin can be used in dogs and cats that are over 6 weeks of age.

Major adverse effects:

May rarely cause topical irritation. Should not be ingested or placed in the eyes.

Fipronil

Trade name: Frontline®.
Formulation: Spray for topical use.

Dose and route of administration:

1. The spray is applied all over the body until the coat is slightly damp. This should equate to a dose rate of 3–6 ml/kg. Animals should not be bathed for 48 hours before or after treatment with fipronil.
2. For the treatment of scabies, cheyletiellosis, notoedric mange and lice, the product should be applied on two occasions a month apart.
3. For the treatment of *Trombicula* infestation, the product should be applied once a month during the appropriate season. Stubbornly infested areas such as the feet may require weekly applications.
4. For the treatment and prevention of tick infestations, fipronil should be applied once a month.
5. The efficacy of fipronil in canine scabies, cheyletiellosis and notoedric mange is variable. Clients must be warned that the clinical signs may persist despite treatment.
6. Frontline spray can be used in dogs and cats from 2 days of age.

Major adverse effects:

May rarely cause topical irritation. Should not be ingested or placed in the eyes. Must not be used in rabbits.

Pyrethroid shampoos (commonly containing permethrin, piperonyl butoxide)

Trade name: Various.
Formulation: Shampoos.

Dose and route of administration:

1. Wet the entire body of the dog with warm water.
2. Apply the shampoo to the skin and lather all over.
3. Allow to soak for 10 minutes.
4. Rinse well and allow to dry.
5. Repeat the bath once a week for 6 weeks.
6. Pyrethroid shampoos can be used in dogs over 12 weeks of age.

Major adverse effects:

Relatively safe but should be used with care in cats. Signs of toxicity include salivation, lethargy, breathing difficulties. If these occur, immediately wash the animal in warm, soapy water.

Selenium sulphide

Trade name: Seleen®, Selsun Blue®.
Formulation: Shampoo.

Dose and route of administration:

1. Wet the entire body of the dog or cat with warm water.
2. Apply the shampoo to the skin and lather all over.
3. Allow to soak for 10 minutes.
4. Rinse well and allow to dry.

5. Repeat the bath once a week for 6 weeks.
6. Selenium sulphide shampoo can be used in dogs and cats of all ages.

Major adverse effects:

Selenium sulphide is poisonous if taken internally so animals should not be allowed to lick the suspension. Veterinary staff and owners should wear protective clothing and gloves when using this product.

Imidacloprid

Trade name: Advantage®.
Formulation: Spot-On for topical use.

Dose and route of administration:

1. Advantage is applied by emptying the contents of an appropriate sized tube onto the skin on the back of the neck in front of the shoulder blades. For large dogs over 40 kg, the product should be applied at more than one place along the dorsal midline.
2. For the treatment of lice, imidacloprid should be applied on two occasions a month apart. Animals should not be bathed after treatment with Advantage. If the animal gets markedly wet, retreatment is necessary.
3. Advantage can be used on dogs and cats that are over 8 weeks of age.

Major adverse effects:
May rarely cause topical irritation. Should not be ingested or placed in the eyes.

Ivermectin

Trade name: Ivomec®.
Formulation: 1% Solution for injection (10 mg/ml).

Dose and route of administration:

1. Ivermectin must never be given to Collies or Collie crosses as it can cause fatal reactions.
2. For the treatment of canine scabies, cheyletiellosis and notoedric mange, ivermectin is administered at a dose of 200 µg/kg (0.2 ml per 10 kg) once a week for 4 weeks. The solution can be given by subcutaneous injection or orally in the dog's food.
3. As a precaution, a pre-treatment test dose can be administered at a dose of 120 µg/kg to identify those dogs that are going to show adverse reactions.

Major adverse effects:

Ivermectin is extremely toxic to Collies, Collie crosses and herding breeds. It should NEVER be given to these breeds. Ivermectin may also produce sporadic toxic reactions in other breeds. Signs of toxicity include mydriasis, depression, tremor, ataxia, stupor, vomiting, salivation, blindness, coma, seizures and death. Most dogs will recover with supportive care (fluid therapy, control of seizures, maintenance of vital functions).

Milbemycin (not available in UK)

Trade name: Interceptor®.
Formulation: 2.3 mg, 5.75 mg, 11.5 mg and 23 mg tablets.

Treatment principles

1. Successful treatment of superficial staphylococcal pyoderma is best achieved with a combination of systemic and topical therapy.
2. For systemic therapy, treat with an appropriate antibiotic (see Table 14.2).
3. Use an adequate dose based on the dog's body weight.
4. Treat for an appropriate length of time. For superficial pyoderma, this will be at least 3 weeks (but at least 1 week past clinical cure).
5. DO NOT USE CONCURRENT CORTICOSTEROIDS, as this will inhibit the body's ability to clear the infection and may lead to recurrence.
6. If possible, treat at the same time with an appropriate shampoo (see later).
7. Treat or control the underlying cause.
8. Reinspect the dog after 3 weeks to ensure clinical resolution. Prescribe further treatment if not completely resolved.

Antibiotics for the treatment of superficial pyoderma

The antibiotics that are suitable for the treatment of superficial pyoderma are listed in Table 14.2 and described below. Antibiotics that are NOT suitable include penicillin, ampicillin, amoxycillin and tetracycline.

Table 14.2 Systemic antibiotics for treating canine superficial pyoderma

Presentation	Drug
First time pyoderma	Clindamycin
	Lincomycin
	Potentiated-sulphonamides (various)
	Tylosin
Recurrent or resistant pyoderma	Cephalexin
	Cephadroxil
	Clavulanic acid potentiated amoxycillin
	Oxacillin
Pyoderma resistant to all antibiotics except fluoroquinolones	Difloxacin
	Enrofloxacin
	Marbofloxacin
	Orbifloxacin

In dogs presenting with superficial pyoderma for the first time, it is appropriate to choose an antibiotic empirically from the list in Table 14.2. Although the clinician can choose any of the antibiotics listed, most dermatologists recommend a staged approach to the use of antibiotics in this condition. This allows for highly effective drugs to be kept in reserve for recurrent or refractory cases. The rationale for this approach is to delay the development of antibiotic resistance by populations of *Staphylococcus intermedius*. The routine use of fluoroquinolones for the treatment of superficial pyoderma is controversial because most dermatologists believe these drugs should be reserved for gram negative, mixed or highly resistant infections.

blood dyscrasias. Potentiated sulphonamides can also lead to a severe euthyroid sick syndrome that can mimic spontaneous hypothyroidism in thyroid function tests.

Ormetoprim potentiated sulphadimethoxine

Trade name: Primor® (not available in UK).
Formulation: 120 mg, 600 mg, and 1200 mg tablets.

Dose and route of administration:

55 mg/kg on day 1, orally
27.5 mg/kg every 24 hours thereafter, orally.

Major adverse effects:

The incidence of keratoconjunctivitis sicca (KCS) is reportedly lower when using this drug compared to trimethoprim potentiated sulphadiazine. However, similar precautions should be taken as described above for trimethoprim potentiated sulphadiazine. Potentiated sulphonamides have also caused immune-mediated polyarthritis in a small number of dogs, especially Doberman pinschers. For this reason, some clinicians avoid the use of potentiated sulphonamides in Dobermans. If polyarthritis occurs, the drug should be stopped and future use avoided in that animal. The clinical signs usually disappear following cessation of therapy. Potentiated sulphonamides can rarely lead to severe, life-threatening adverse drug reactions such as erythema multiforme, toxic epidermal necrolysis or blood dyscrasias. Potentiated sulphonamides can also lead to a severe euthyroid sick syndrome that can mimic spontaneous hypothyroidism in thyroid function tests.

Bacquiloprim potentiated sulphadimethoxine

Trade name: Zacquilan®.
Formulation: 60 mg and 600 mg tablets.

Dose and route of administration:

30 mg/kg every 48 hours, orally.

Major adverse effects:

The incidence of keratoconjunctivitis sicca (KCS) is reportedly lower when using this drug compared to trimethoprim potentiated sulphadiazine. However, similar precautions should be taken as described above for trimethoprim potentiated sulphadiazine. Potentiated sulphonamides have also caused immune-mediated polyarthritis in a small number of dogs, especially Doberman pinschers. For this reason, some clinicians avoid the use of potentiated sulphonamides in Dobermans. If polyarthritis occurs, the drug should be stopped and future use avoided in that animal. The clinical signs usually disappear following cessation of therapy. Potentiated sulphonamides can rarely lead to severe, life-threatening adverse drug reactions such as erythema multiforme, toxic epidermal necrolysis or blood dyscrasias. Potentiated sulphonamides can also lead to a severe euthyroid sick syndrome that can mimic spontaneous hypothyroidism in dynamic thyroid function tests. Bacquiloprim potentiated sulphadimethoxine should be avoided in dogs with severe liver disease.

Tylosin

Trade name: Tylacare®.
Formulation: 200 mg tablets.

Dose and route of administration:

10–20 mg/kg every 12 hours, orally.

Major adverse effects:

None reported.

Cephalexin

Trade names: Ceporex®, Keflex®, Rilexine®.
Formulation: 50 mg, 250 mg, 500 mg, 75 mg, 300 mg, 600 mg tablets, 100 mg/ml oral suspension, depending on the manufacturer.

Dose and route of administration:

22–30 mg/kg every 12 hours, orally.

Major adverse effects:

Hypersensitivity to cephalexin is very rare. The drug should not be given to dogs, or handled by people, known to be allergic to penicillin or cephalosporins. The dose of cephalexin should be reduced in dogs with pre-existing renal disease. Oral administration of cephalexin often causes vomiting in dogs but this usually subsides after the first few doses. The problem is reduced if the drug is given with food.

Cephadroxil

Trade name: Cefa-Tabs®.
Formulation: 50 mg, 100 mg and 200 mg tablets.

Dose and route of administration:

22 mg/kg every 12 hours, orally.

Major adverse effects:

Hypersensitivity to cephadroxil is very rare. The drug should not be given to dogs, or handled by people, known to be allergic to penicillin or cephalosporins. The dose of cephadroxil should be reduced in dogs with pre-existing renal disease. Oral administration of cephadroxil may cause vomiting in dogs but this usually subsides after the first few doses. The problem is reduced if the drug is given with food.

Clavulanic acid potentiated amoxycillin

Trade names: Clavamox®, Synulox®.
Formulation: 50 mg, 62.5 mg, 125 mg, 250 mg, 375 mg, 500 mg tablets; 50 mg/ml oral suspension.

Dose and route of administration:

12–15 mg/kg every 12 hours, orally.

Major adverse effects:

Hypersensitivity to amoxycillin can occur. The drug should not be given to dogs, or handled by people, known to be allergic to penicillins or cephalosporins.

Oxacillin

Trade name: Generic (not available in the UK).
Formulation: 250 mg capsule.

Dose and route of administration:

22 mg/kg every 8 hours, orally.

Major adverse effects:

Hypersensitivity to oxacillin can occur. The drug should not be given to dogs, or handled by people, known to be allergic to penicillins or cephalosporins. The drug should not be administered with food.

Difloxacin

Trade name: Dicural®.
Formulation: 15 mg, 50 mg and 100 mg tablets.

Dose and route of administration:

5 mg/kg every 24 hours, orally.

Major adverse effects:

Difloxacin should not be administered to rapidly growing dogs as it can cause damage to weight-bearing articular cartilage. This period includes up to 8 months in small dogs, 12 months in medium dogs and 18 months in large dogs. Difloxacin may occasionally cause vomiting and rarely cause inappetence, diarrhoea and anal irritation. The concurrent use of fluoroquinolones and non-steroidal anti-inflammatory drugs should be avoided in epileptic patients.

Enrofloxacin

Trade name: Baytril®.
Formulation: 15 mg, 50 mg and 150 mg tablets.

Dose and route of administration:

5 mg/kg every 24 hours, orally.

Major adverse effects:

Enrofloxacin should not be administered to rapidly growing dogs as it can cause damage to weight-bearing articular cartilage. This period includes up to 8 months in small dogs, 12 months in medium dogs and 18 months in large dogs.

Marbofloxacin

Trade name: Marbocyl®.
Formulation: 5 mg, 20 mg and 80 mg tablets.

Dose and route of administration:

2 mg/kg every 24 hours, orally.

Major adverse effects:

Marbofloxacin should not be administered to rapidly growing dogs as it can cause damage to weight-bearing articular cartilage. This period includes up to 8 months in small dogs, 12 months in medium dogs and 18 months in large dogs. Marbofloxacin can occasionally cause vomiting, diarrhoea, modification of thirst and changes in activity. Overdosage with marbofloxacin can cause neurological disorders and the drug should be used with caution in epileptic patients.

What if the pyoderma responds but then relapses?

Many cases of superficial pyoderma respond to appropriate antibacterial therapy and do not recur. In these cases, the underlying cause is likely to be something transient such as swimming in dirty water; lying on dirty ground; trauma to the skin from thorns or grooming; transient scratching or licking; or an immature immune system. In some cases, the original cause of the infection is never established.

In other cases, the infection responds completely to antibacterial therapy but then relapses a few days to a few weeks after the therapy has been stopped. The most common reason for this is failure to identify and treat an underlying cause. In such cases, clinicians should rule out:

- Ectoparasitism, especially demodicosis
- Atopic dermatitis
- Food allergy
- Hypothyroidism
- Hyperadrenocorticism
- Sex hormone abnormality
- Systemic diseases
- Primary scaling disorders

The order in which these underlying causes are investigated should be based on the clinician's index of suspicion. If the condition is still pruritic after the bacterial infection has resolved, the parasitic and allergic diseases should be ruled out first. If the condition is non-pruritic after the infection has resolved, the most likely causes are sub-clinical atopic dermatitis, demodicosis, hypothyroidism, hyperadrenocorticism, sex hormone abnormalities and systemic diseases.

If all the above conditions have been ruled out and the infection keeps recurring, the clinician can make a diagnosis of idiopathic recurrent pyoderma. This condition is thought to be due to a cutaneous immunodeficiency but there are currently no diagnostic tests that can be used to confirm the diagnosis. In such cases, there are three therapeutic strategies that can be used for long term maintenance.

1. Aggressive and frequent topical therapy with one of the shampoos described above. Although this strategy is beneficial, it rarely controls the pyoderma alone.
2. Immunomodulation with staphylococcal bacterins. Some external laboratories can make an autogenous bacterin from staphylococcal organisms cultured from the affected dog. Alternatively, a commercially available bacterin such as Staphage lysate® can be considered. The success rate of both these strategies is variable, but they do represent an alternative to continuous antibiotic therapy in some cases.
3. Long-term antibiotic therapy. After the pyoderma has been brought under control with antibiotics, a reducing dosage regime can be prescribed. This may take the form of pulse therapy (e.g. 1 week on, 2 weeks off), or daily low dose therapy. This type of regime has to be tailored to the individual case taking into account such factors as the frequency of relapse and variation in response. For long-term use, cephalexin, oxacillin or clavulanic acid potentiated amoxycillin are the antibiotics of choice.

As the need for this type of therapy is rare, clinicians are advised to seek further advice from dermatology specialists before embarking on these treatment regimes.

Dose and route of administration:

Topical application as described above

Major adverse effects:

Malaseb® can be irritating to the eyes and contact with the dog's and owner's eyes should be avoided. In case of accidental contact, the eyes should be rinsed with plenty of water. Malaseb® can also occasionally cause skin irritation.

2% Miconazole (shampoo formulation not available in UK)

Trade name: Daktarin cream®, Daktarin spray®, Conofite cream®, Miconazole shampoo®, Miconazole spray®.
Formulation: Cream, spray or shampoo.

Dose and route of administration:

Topical application of shampoo as described above. Creams and sprays are applied directly to affected areas once daily initially and then as necessary.

Major adverse effects:

Miconazole can be irritating to the eyes and contact with the dog's and owner's eyes should be avoided. In case of accidental contact, the eyes should be rinsed with plenty of water.

2% acetic acid/2% boric acid (not available in UK)

Trade name: MalAcetic®.
Formulation: Shampoo or wet wipes.

Dose and route of administration:

Topical application of shampoo as described above. Wet wipes are used to clean the skin directly.

Major adverse effects:

None reported.

2% Ketoconazole

Trade name: Nizoral®.
Formulation: Shampoo.

Dose and route of administration:

Topical application as described above.

Major adverse effects:

Not licensed for use in animals.

Systemic treatment of *Malassezia* dermatitis

Ketoconazole

Trade name: Nizoral®.
Formulation: 200 mg tablet.

- Allergen immunotherapy
- Allergen avoidance
- Essential fatty acids
- Antihistamines
- Topical therapy
- Glucocorticoids
- Other immunomodulatory drugs.

Allergen immunotherapy

Allergen immunotherapy (hyposensitisation or desensitisation) involves the subcutaneous injection of gradually increasing doses of relevant allergens over a period of time until a maintenance dose is reached. The treatment is thought to work by correcting the imbalance in the animal's T lymphocyte populations with the net effect that Interleukin-4 secretion is reduced. This reduces IgE synthesis and encourages IgG synthesis, which may be protective.

Formulation, dosage schedule and route of administration:

Selection of the allergens to be included in the vaccine is based on the positive results obtained in either the intradermal skin test or in-vitro test. Some companies and dermatologists limit the number of allergens in a vaccine to ten. If more than ten allergens are thought to be significant, a second vaccine can be made up. Other dermatologists simply put all the relevant allergens into one vaccine. To date, there is no evidence that one method is superior to the other.

Allergen vaccines are either prepared in aqueous form or alum-precipitated form. Both types are given subcutaneously, although alum-precipitated vaccines should be injected at four different sites around the animal to avoid reactions (right neck, left neck, right flank, left flank).

Aqueous vaccines are prepared in three different vials, with vial 3 being ten times more concentrated than vial 2, and vial 2 being ten times more concentrated than vial 1. Alum-precipitated vaccines are prepared in a single vial.

The dosage schedules for these two formulations are different because of different rates of absorption. The schedule for aqueous allergens is shown in Table 14.3 and alum-precipitated allergens in Table 14.4.

Efficacy

Allergen immunotherapy takes 2–9 months before a beneficial effect is seen. The normal recommendation is that dogs receive the vaccine for at least a year before a final decision is made as to its efficacy. Therefore, during the early phase of allergen immunotherapy, it is usually necessary to use some additional therapy to control the clinical signs. In most studies published to date, similar success rates have been reported. Twenty-five per cent of atopic dogs can be controlled with immunotherapy alone. A further 50% show a beneficial response, but the dog requires some other form of non-steroidal therapy. The remaining 25% show no response at all. The success rate of allergen immunotherapy in cats is suspected to be similar, but precise figures are not known. Owners need to be informed of the slow nature of this type of treatment and the likely success rates so that they don't have unrealistic expectations. If successful, allergen immunotherapy must be continued for the remainder of the animal's life in the vast majority of cases.

Table 14.3 Injection schedule for aqueous allergens

Day	Vial	Dose (ml)
1	1	0.1
2	1	0.2
4	1	0.4
6	1	0.8
8	1	1.0
10	2	0.1
12	2	0.2
14	2	0.4
16	2	0.8
18	2	1.0
20	3	0.1
22	3	0.2
24	3	0.4
26	3	0.8
28	3	1.0
38	3	1.0
48	3	1.0

Thereafter, 1.0 ml every 20 to 40 days as required

Table 14.4 Injection schedule for alum-precipitated allergens

Week	Day	Dose (ml)
0	0	0.2
2	14	0.4
4	28	0.6
6	42	0.8
9	63	1.0
12	84	1.0
16	112	1.0
20	140	1.0

Thereafter, 1.0 ml per month

Adverse effects:

Adverse effects of allergen immunotherapy are uncommon. However, as a precaution, the first five injections of the course should be given under veterinary supervision and the animal should remain in the clinic for 30 minutes to monitor for any adverse reactions. Any problems are likely to occur during this time. Potential adverse effects and the recommended course of action are shown in Table 14.5. If no problems occur, further injections can be administered at home by the owner (following appropriate instruction).

Table 14.5 Adverse effects of allergen immunotherapy

Adverse effect	Treatment
Mild pruritus at the injection site	None
Generalised pruritus	Administer antihistamines for 2 days before the next injection
Generalised urticaria	Administer 1–2 mg/kg diphenhydramine IM Administer glucocorticoids by injection Administer antihistamines for 2 days before the next injection
Anaphylaxis (vomiting, diarrhoea, pale mucous membranes, respiratory distress, collapse)	Administer 0.01 ml/kg 1:1000 epinephrine IM or SC Administer 1–2 mg/kg diphenhydramine IM Administer glucocorticoids by injection Monitor very carefully if the injection course is continued Provide owner with an EpiPen® (automatic epinephrine injector available from pharmacists)

What if the treatment fails?

Animals may fail to respond to allergen immunotherapy for the following reasons:

Reason for failure	*Solution*
Animal improves after each injection but relapses before the next one is due	Give injections more frequently
Animal improves initially but response seems to deteriorate as the course progresses	Check for concurrent problems. If none are present, reduce dose of injection by 50%. If still no improvement, reduce dose by a further 50%. If still no improvement, repeat allergy tests and food trials. If additional allergens are identified, modify vaccine prescription
Animal is doing well but becomes more pruritic after each injection	Reduce dose of injections by 50%
Animal completely fails to respond despite taking above measures	25–30% of dogs will fail to respond to allergen immunotherapy. Discontinue and resort to other therapies

Allergen avoidance

Although total allergen avoidance would be the ideal method of controlling atopic dermatitis, it is difficult to achieve in practice. The best way of approaching this subject is to give the client an advice sheet and recommend that they attempt as much as is feasibly possible (Table 14.6).

Essential fatty acids

Essential fatty acids are usually derived from evening primrose oil, borage oil and marine fish oil. The rationale for the use of essential fatty acids in the treatment of atopic dermatitis is based on their ability to modify and reduce the production of pro-inflammatory prostaglandins in the skin.

Trade names and formulations:

Viacutan® (UK) – pump canister, capsules; EFADOSE® (Europe) – liquid; Derm Caps® (USA) – capsules, liquid.

Table 14.6 Strategies for allergen avoidance

Allergen	Suggestions for avoidance
House dust and dust mites	Keep pet out of bedroom Use filtered vacuum cleaner Use hypoallergenic mattress and pillow covers Avoid stuffed toys – or place them in deep freeze for 24 hours once a month Wash bedding in hot water (>70°C) Use anti-dust mite sprays (environmental flea control products with pyrethroids are satisfactory)
Pollens	Find out the plants and trees that are present in the dog's location (a good investment for clients is a plant identification book for grasses, wild flowers and trees) Try to avoid areas during the pollination season (Trees – Spring; Grasses – Summer; Weeds – Autumn) Keep lawns mowed short Rinse dog off after walking through vegetation

Dose and route of administration:

To have an anti-pruritic effect, essential fatty acids should be administered at two to four times the dose recommended on the package. In addition, they need to be given for 6 to 8 weeks for maximum effect.

Efficacy:

At best, essential fatty acids are only likely to be beneficial in about 10–20% of cases. When combined with antihistamines, there can be a synergistic effect. Even if there is no effect on the level of pruritus, essential fatty acid supplementation often leads to a noticeable improvement in skin and coat quality.

Major adverse effects:

Essential fatty acids can occasionally cause diarrhoea. Rarely, essential fatty acids have precipitated pancreatitis.

What if the treatment fails?

Animals may fail to respond to essential fatty acid supplementation for the following reasons:

Reason for failure	*Solution*
Underdosing	Ensure the dose is high enough for the animal's body weight
Insufficient duration of therapy	Treat for a minimum of 6 to 8 weeks
Animal has concurrent problems	Completely eliminate parasites, infections and food allergy before using essential fatty acids
Animal completely fails to respond despite taking above measures	80–85% of dogs will fail to respond to essential fatty acids. Discontinue and resort to other therapies

Antihistamines

Antihistamines are used in the management of atopic dermatitis because histamine is a major inflammatory mediator released from mast cells during allergic inflammation.

The following antihistamines are commonly used to treat canine atopic dermatitis.

Chlorpheniramine

Trade name: Piriton®.
Formulation: 4 mg tablet.

Dose and route of administration:

0.4 mg/kg every 8 hours, orally.

Clemastine

Trade names: Tavegil®, Tavist®, Aller-eze®.
Formulation: 1 mg tablet.

Dose and route of administration:

0.05 mg/kg every 12 hours, orally.

Hydroxyzine

Trade names: Atarax®.
Formulation: 25 mg tablet.

Dose and route of administration:

2.2 mg/kg every 8 hours, orally.

Diphenhydramine

Trade names: Benadryl®, Nytol®.
Formulation: 25 mg tablet.

Dose and route of administration:

2.2 mg/kg every 8 hours, orally.

Cyproheptadine

Trade names: Periactin®.
Formulation: 4 mg tablet.

Dose and route of administration:

0.1–1.0 mg/kg every 12 hours, orally.

Efficacy

Antihistamines have been found to be ineffective for the treatment of atopic dermatitis by many practitioners. However, although they cannot be regarded as a substitute for glucocorticoids, they can be effective in 10–20% of cases. It is important when prescribing antihistamines to try at least three different drugs for a week each before determining the overall efficacy. This is because failure to respond to one drug does not imply that the animal will not respond to another. The antihistamine trial should only be attempted when all other secondary problems are under control. The client should be told to keep a diary and note the level of pruritus each week. If one drug is clearly better than the others, it should be repeated to determine if the effect is sustained. If it is, the drug can be continued in the medium to long term, or used as short-term palliative treatment during the early stages of allergen immunotherapy.

Major adverse effects:

Antihistamines are not licensed for the treatment of animals in the UK or USA. The major adverse effect of antihistamines is drowsiness. If this occurs, the drug should be stopped and an alternative antihistamine tried.

What if the treatment fails?

Animals may fail to respond to antihistamines for the following reasons:

Reason for failure	*Solution*
Only one drug tried	Try at least three different types before concluding that antihistamines are ineffective
Animal has concurrent problems	Completely eliminate parasites, infections and food allergy before using antihistamines
Animal completely fails to respond despite taking above measures	80–90% of dogs will fail to respond to antihistamines. Discontinue and resort to other therapies

Topical therapy

Topical therapy is an invaluable part of the management programme when treating canine atopic dermatitis but it is rarely used in cats. Topical therapy may take the form of shampoos, soaks, sprays or ointments. Shampoos are beneficial because:

- They can be used to manage secondary bacterial or yeast infections.
- They remove sweat, sebum and other cutaneous debris.
- They can be used primarily for anti-pruritic effects.
- They can remove allergens from the skin surface.

In addition to antimicrobial shampoos (described in earlier sections), the following shampoos and soaks can be useful for topical treatment in atopic dermatitis. The ointments and sprays can be used for spot treatment of focal areas of the body that are proving difficult to control (e.g. the perineum, the feet, the medial pinnae).

When using shampoos, the following basic procedure should be followed:

1. Wet the entire body of the dog with warm water.
2. Apply small quantities of shampoo to various parts of the body and lather all over. Avoid getting shampoo into the eyes.
3. Massage the shampoo into the skin for 10 minutes, especially in the most severely affected areas. The 10-minute contact time is essential for therapeutic activity.
4. Rinse well and allow to dry in a warm, draught-free environment.
5. Repeat the bath two or three times a week initially, and then as required.

Colloidal oatmeal

Trade name: Episoothe®.
Formulation: Shampoo and soak.

Beneficial and adverse effects:

The value of topically applied essential fatty acids has not been proven although they are intended to have anti-pruritic and coat conditioning effects. No major adverse effects have been reported.

Topical glucocorticoids

Trade names: Hydrocortisone cream, Betsolan cream®, Corticalm®, Cortispray®, Dermacool HC®, Fuciderm®, Hydro-10 mist®, Hydro-Plus®, Panalog®, PTD-HC®, Tresaderm®, Vetalog®, Vetsovate® (not all available in UK or USA).
Formulations: Sprays, creams.

Dose and route of administration:

Topical glucocorticoids vary in potency. Betamethasone and dexamethasone are the most potent, followed by triamcinolone, followed by hydrocortisone. Clinicians should decide on the level of potency required and apply the creams or sprays once or twice daily to the affected area. If prolonged treatment is used, the lowest potency cream should be used at the lowest frequency possible.

Beneficial and adverse effects:

Topical glucocorticoids are potent anti-inflammatory drugs and they can be very effective at treating focal areas of atopic dermatitis. However, topical glucocorticoids can suppress the pituitary adrenal axis and lead to elevated liver enzymes. Prolonged use can also cause thinning of the skin, scaling, comedones, alopecia and secondary pyoderma. Owners should wear gloves when applying these products.

Hamamelis extract and menthol

Trade name: Dermacool®.
Formulation: Spray.

Dose and route of administration:

Spray onto the affected area.

Beneficial and adverse effects:

This spray has a cooling, soothing, and astringent action although the effects are transient and appear to decrease with repetitive use. No adverse effects have been reported.

Systemic glucocorticoids

Systemic glucocorticoids are indicated for the treatment of atopic dermatitis in the following circumstances:

- In cases of seasonal atopic dermatitis lasting 3 months or less. In these cases, glucocorticoids are the treatment of choice as long as they are well tolerated by the patient.
- In the early phases of allergen immunotherapy if the pruritus is severe.
- As an adjunctive treatment in cases that fail to completely respond to other treatment modalities.
- As a sole treatment in cases that have not responded at all to other treatment modalities.
- In cases in which financial limitations do not permit other forms of treatment.

- In cases in which owners are not prepared to pursue other forms of treatment.

Oral prednisolone (or prednisone) is the drug of choice for the treatment of atopic dermatitis. In the vast majority of cases, there is no indication for using any other drug. In cases in which severe polydipsia and polyuria are a problem, oral methyl prednisolone can be used as an alternative. Other oral steroids such as triamcinolone, betamethasone and dexamethasone are less suitable for the treatment of atopic dermatitis because their long duration of action does not allow successful alternate day therapy. Injectable steroids should be avoided because they do not allow precise dosage control. They should only be contemplated in cases in which the owner is totally unable to administer tablets to their animal.

Prednisolone

Trade names: Prednicare®, Prednidale 5®, Prednisolone tablets B.P.®, Precortisyl®.
Formulation: 1 mg, 5 mg and 25 mg tablets, depending on the manufacturer.

Dose and route of administration:

For dogs, 0.5–1.0 mg/kg/day divided into two doses given 12 hours apart. After a beneficial response is seen (5–7 days), the daily dose can be given every 24 hours for a further 3–5 days. After that, the total dose can be given every 48 hours. For long-term use, the alternate day therapy should be gradually reduced to the lowest dose that is capable of controlling the pruritus. For cats, these doses should be doubled.

Methylprednisolone

Trade name: Medrone V®, Medrol®.
Formulation: 2 mg and 4 mg tablets.

Dose and route of administration:

For dogs, 0.4–0.8 mg/kg/day divided into two doses given 12 hours apart. After a beneficial response is seen (5–7 days), the daily dose can be given every 24 hours for a further 3–5 days. After that, the total dose can be given every second day. For long-term use, the alternate day therapy should be gradually reduced to the lowest dose that is capable of controlling the pruritus. For cats, these doses should be doubled.

Methylprednisolone acetate

Trade name: Depo-Medrone®, Depo-Medrol®.
Formulation: 40 mg/ml suspension.

Dose and route of administration:

For dogs, 1–2 mg/kg by intramuscular injection.
For cats, 5 mg/kg by intramuscular injection (approximately 20 mg per cat).
The effects can last for up to 4 weeks.

In dogs, the drug should only be used if all other alternatives have been explored because atopic dermatitis will require life-long injections and major side effects are likely to occur. Cats are more tolerant to the drug.

Efficacy of glucocorticoids:

Systemic glucocorticoids are highly effective at controlling the pruritus in atopic dermatitis.

Major adverse effects of glucocorticoids:

The major drawback to the use of systemic glucocorticoids in dogs is the large number of adverse effects that will invariably occur. Short-term use of systemic glucocorticoids can lead to polydipsia, polyuria, polyphagia, panting, aggression and diarrhoea. Methylprednisolone has fewer mineralocorticoid effects than prednisolone and can reduce the severity of polydipsia and polyuria. Prolonged use of glucocorticoids can lead to signs of iatrogenic hyperglucocorticism (muscle wastage, pot belly, hepatomegaly, fat redistribution, osteoporosis, calcinosis cutis, alopecia, poor wound healing, recurrent pyoderma, generalised demodicosis, comedones, silent urinary tract infections, pyelonephritis, cataracts, insulin resistant diabetes mellitus). Despite requiring higher doses of glucocorticoids, these adverse effects are rare in cats. Glucocorticoids have caused gastro-intestinal ulceration, especially in patients receiving non-steroidal anti-inflammatory drugs. Glucocorticoids may also predispose to pancreatitis. Sudden withdrawal of glucocorticoids after prolonged therapy can lead to an Addisonian crisis (adrenal insufficiency). Glucocorticoids should be avoided in animals with pre-existing renal disease and diabetes mellitus. Glucocorticoids should not be given to pregnant animals as they can cause foetal abnormalities and abortion.

What if the treatment fails?

Animals may fail to respond to glucocorticoids for the following reasons:

Reason for failure	*Solution*
Animal has concurrent problems	Completely eliminate parasites, infections and food allergy before concluding that glucocorticoids are ineffective
Underdosing	Ensure that an appropriate dose is used initially
Incorrect diagnosis	Rule out other causes of pruritus such as epitheliotropic lymphoma

Other imunomodulatory drugs

Various other immunomodulatory drugs and treatments have been investigated for their efficacy in atopic dermatitis. In the future, some of these treatments (and others) may become licensed, more cost effective and widely available. These treatments are summarised in Table 14.7.

Table 14.7 Immunomodulatory drugs for the treatment of atopic dermatitis

Drug	Dose	Efficacy	Disadvantages
Cyclosporine	5–10 mg/kg/day	Good	Very expensive
Misoprostol	6 µg/kg every 8 hours	Fair	Not widely available
Pentoxifylline	10 mg/kg every 8 hours	Poor	Poor efficacy
Arofylline	1 mg/kg every 12 hours	Good	Causes vomiting, not widely available
Chinese herbs	As directed	Fair	Not yet available

Devising a long-term management programme

All the treatment modalities described in this section are merely tools to be used in the management of atopic dermatitis. They need to be put together in a way that allows successful control of the disease over a prolonged period of time. The following strategy represents a typical plan that might be recommended for an atopic dog.

loma complex, a course of glucocorticoids may be indicated. This will provide immediate symptomatic relief for the patient and may encourage the client to pursue what seems initially like a long investigation. Symptomatic treatment of feline symmetrical alopecia is not usually necessary because the skin is not inflamed and the severity of the condition is less than that of the other patterns.

4. Prednisolone should be prescribed at an initial dose of 2 mg/kg/day, divided into two doses given 12 hours apart. This initial dose should last for 5–14 days, depending on the severity and response. Lesions of the eosinophilic granuloma complex and head and neck pruritus occasionally require higher doses than this.
5. Once the lesions have responded, the dose should be tapered and stopped. This allows the effects of specific treatments to be determined e.g. response to flea control, ectoparasiticides, food trials, etc.
6. Long-term depot injections of glucocorticoids should be avoided because they may interfere with future testing (especially intradermal testing).
7. If there is cytological evidence of bacterial infection (especially eosinophilic granuloma complex), systemic antibiotics should be prescribed for 3–4 weeks.
8. If dermatophytosis is suspected (especially symmetrical alopecia), avoid the use of glucocorticoids until the condition has been ruled in or out on culture.
9. Glucocorticoids must be withdrawn for 3–4 weeks before intradermal testing (depending on the dose/duration of therapy). Clinicians should check with the testing site what is required.

Treatment principles for chronic or idiopathic cases

1. If the diagnostic work-up does not yield a definitive diagnosis, long-term management may be required.
2. Prednisolone should be used on an alternate day basis at a dose of 1–2 mg/kg/day.
3. If the lesions do not respond, or if the owner can not administer tablets on a long-term basis, consider depot injections of methylprednisolone acetate (20 mg/cat) every 2 months.
4. Try antihistamines to determine if the dose of steroids can be reduced. Chlorpheniramine should be prescribed at a dose of 2 mg/cat every 12 hours.
5. Try essential fatty acids to determine if the dose of steroids can be reduced. Prescribe an appropriate product at twice the recommended dose.
6. For indolent lip ulcers, antibacterial therapy or surgical excision may be curative if other treatments have failed.
7. As a last resort, the use of megestrol acetate can be considered for the treatment of feline cutaneous reaction patterns.

Products for the treatment of feline cutaneous reaction patterns

The majority of cases of feline cutaneous reaction patterns can be successfully managed using drugs described in earlier sections of this chapter. Readers should consult the sections on fleas, superficial ectoparasites, bacterial infections, food allergy and atopic dermatitis. Clinicians should only use the following drug if the previous treatments have been ineffective.

Megestrol acetate

Trade name: Ovarid®, Ovaban®
Formulation: 5 mg and 20 mg tablets.

Dose and route of administration:

5 mg per cat every other day, orally, until the lesions have begun to regress. Then, 2.5–5 mg per cat once a week.

Major adverse effects:

Megestrol acetate can cause diabetes mellitus, mammary hyperplasia, mammary neoplasia, temperament changes, polydipsia, polyuria and pyometritis.

TREATMENT OF HOT SPOTS (PYOTRAUMATIC DERMATITIS)

Treatment objectives

1. To identify and treat the underlying cause.
2. To provide symptomatic treatment of the hot spot itself.

Treatment principles

1. Perform appropriate tests to determine the underlying cause (see Chapters 3 and 13). Initiate specific treatment as appropriate.
2. Check the anal sacs or ears (depending on the location of the lesion). Express the anal sacs if necessary and treat the ears if abnormal.
3. Clip away matted fur from around the lesion. Check to see if there is evidence of superficial pyoderma in the surrounding skin. If there is, initiate appropriate treatment (see, p. 240).
4. Clean the lesion and remove crusted exudate using a dilute solution of chlorhexidene or povidone-iodine.
5. In most cases, treatment with glucocorticoids is indicated to break the itch-scratch cycle. Oral prednisolone can be administered at a dose of 1 mg/kg daily for 3–5 days followed by 2 days at 0.5 mg/kg.
6. Topical application of antibiotic/glucocorticoid cream is also beneficial to provide a localised anti-pruritic effect and to treat the surface bacterial colonisation.
7. Other topical anti-pruritic agents can be used in less severe cases.

Products for the treatment of hot spots

The use of systemic antibiotics and systemic glucocorticoids has been described in previous sections of this chapter. Topical products suitable for applying to hot spots are described below.

Topical Antibiotic/Glucocorticoid creams

Trade names: Betsolan cream®, Dermobion®, Fuciderm®, Panalog®, Tresaderm®, Vetodale®, Vetsovate® (not all available in UK or USA).

Dose and route of administration:

Apply to the affected area twice daily initially until the lesion begins to regress. Then apply once daily until completely healed.

Beneficial and adverse effects:

Topical glucocorticoids can rapidly alleviate the intense pruritus associated with hot spots. However, they can suppress the pituitary adrenal axis and lead to elevated liver enzymes.

antibiotics. Cephalexin is the drug of choice and treatment must be continued for up to 8 weeks. The lesion should also be washed daily with benzoyl peroxide shampoo, unless this proves to be irritant.

5. If the clinician is sure that infection is not involved, or if the lesion fails to respond completely to antibacterial therapy, various topical therapies have been recommended that can be applied to the lesion directly. The recipes for these treatments are described below.
6. If the lesion does not respond to topical treatment, the clinician can try sublesional injections of methylprednisolone acetate (Depo-Medrone®, Depo-Medrol®). This is only likely to be successful in small lesions less than 3 cm in diameter.
7. If the clinician suspects a psychological component to the problem, behaviour modification is indicated. This might include the provision of more walks, more freedom, more human companionship, more canine companionship, more toys, more chews, and removal of obvious sources of stress.
8. If behaviour modification is not possible or not totally successful, various psychological drugs can be prescribed. These include clomipramine, fluoxetine, amitriptyline, doxepin or megestrol acetate.
9. If medical management fails, other therapies that have been performed with variable success include surgical resection, laser therapy, radiation therapy, cryosurgery, and acupuncture.

Topical products for the treatment of acral lick dermatitis

Specific treatments for the underlying causes that may lead to acral lick dermatitis are described in other sections of this chapter. Additional topical therapies that are useful in this condition are described below.

Topical mixture 1 (suitable for UK) – Propylene glycol/malic acid/benzoic acid/salicylic acid/enrofloxacin/dexamethasone/DMSO mixture

Trade name: Contains Dermisol solution®, Baytril®, Dexamethasone and DMSO.
Formulation: Home-made solution.

Dose and route of administration:

The mixture is made by adding 2 ml of 2.5% injectable Baytril®, 2 mg of injectable dexamethasone phosphate, and 1 ml of medical grade DMSO to 12 ml of Dermisol Multicleanse Solution®. The solution is applied topically to the lesion twice daily using a paintbrush until completely healed.

Topical mixture 2 (suitable for USA) – Fluocinolone/DMSO/flunixin meglumine mixture

Trade name: Contains Synotic® and Banamine®.
Formulation: Home-made solution.

Dose and route of administration:

The mixture is made by adding 3 ml of Banamine® to an 8 ml vial of Synotic®. The solution is applied topically to the lesion twice daily using a paintbrush until completely healed.

Major adverse effects of topical mixtures 1 and 2:

Owners should wear gloves when applying these mixtures. Dogs must not be allowed to lick the solutions off. Prolonged use of topical glucocorticoids can suppress the pituitary

Major adverse effects of psychological drug therapy:

These drugs should not be used in male breeding dogs or pregnant females. All these drugs can cause anorexia, vomiting, diarrhoea, lethargy, hyperexcitability, polydipsia, personality changes and reduction in seizure threshold. More serious adverse effects include cardiac arrhythmias, heart block, dry mouth, urine retention and reduced tear production. These drugs should be used with caution in dogs with cardiovascular disease, epilepsy, glaucoma, reduced gastrointestinal motility and urinary retention. Massive overdosage with these drugs can cause cardiac dysrhythmias, neurological signs and death.

What if the treatment fails?

Acral lick dermatitis may fail to respond to treatment for the following reasons:

Reason for failure	*Solution*
Underlying cause not identified	Try and determine the underlying cause
Presence of infection not recognised	Treat with long course of systemic antibiotics
Lesion is caused by neurological compression that could not be identified (e.g. neural or perineural tumour)	None, until lesion becomes apparent
Dog's behaviour cannot be modified	None, if all therapeutic options have been explored
Incorrect diagnosis	Consider other differentials (neoplasia, kerion)

TREATMENT OF DEMODICOSIS

Treatment objectives

1. To eliminate the excessive number of demodex mites.
2. To attempt to identify and treat the underlying cause in adult-onset cases.

Treatment principles

1. The treatment recommendations for demodicosis vary depending on whether the case is localised or generalised.
2. With localised demodicosis, 90% of cases will resolve spontaneously. The most appropriate course of action when presented with a case for the first time is to discuss the condition with the owner, delay treatment and arrange a reinspection 4 weeks later. If the condition is resolving or completely static, no treatment is indicated. However, if there is any evidence of new lesions, or the original lesions are spreading, the condition should be treated as for generalised demodicosis.
3. The treatment of generalised demodicosis can be a long, frustrating and expensive process. If a successful outcome is to be expected, it is important to pay close attention to the following treatment guidelines.
4. NEVER ADMINISTER GLUCOCORTICOIDS TO A DOG WITH DEMODICOSIS, EVEN IF IT IS HIGHLY PRURITIC.
5. If the disease develops when the dog is an adult (over 2 years of age), try and identify an underlying cause. Perform routine haematology and biochemistry, urine analysis and rule out hyperadrenocorticism. In addition, thoracic and abdominal radiography and ultrasonography may be indicated.
6. If there is secondary bacterial infection, the dog should be treated concurrently with systemic antibiotics until the pyoderma has resolved.

7. Dogs with medium or long hair coats should be clipped to allow effective miticidal treatment.
8. Prior to each miticidal treatment, the dog should be bathed in a benzoyl peroxide shampoo. This removes scale and crusts, opens up the follicles, and kills bacteria. If the benzoyl peroxide proves irritant, an alternative antibacterial shampoo can be used. The dog should be towel dried before the miticidal treatment is applied to avoid dilution.
9. Miticidal treatment should be commenced initially with a licensed product. In the UK and USA, the only licensed product for the treatment of demodicosis is amitraz. Initial use of other products in the UK contravenes the prescribing cascade. Initial use of other products in the USA is a violation of federal law. Amitraz should be applied at the appropriate concentration at the appropriate intervals (see below).
10. The dog should be re-examined every 4 weeks. Take five skin scrapings from different body regions at each visit. When a set of completely negative skin scrapings is obtained, repeat the scrapings 4 weeks later. If that set is also negative, the treatment can be discontinued (see Chapter 13 for details on performing and interpreting skin scrapings). However, the dog should be monitored carefully for relapses.
11. Three to nine months of continuous treatment may be required to achieve parasitological cure. However, during this time, the dog should be making continuous clinical progress and the mite numbers should be decreasing on skin scrapings.
12. Entire bitches should be spayed when the disease has been brought under control but before treatment is stopped. This is to prevent the disease relapsing when the dog comes into oestrus.
13. There are no licensed products for the treatment of feline demodicosis. Recommended treatments include lime sulfur dips and selenium sulphide shampoos (see the section on superficial ectoparasites).

Products for the treatment of demodicosis

Amitraz wash

Trade names: Aludex® (UK), Mitaban® (USA).
Formulation: Concentrated liquid for dilution and topical use.

Dose and route of administration:

1. Owners and veterinary staff should wear gloves and protective clothing when using this product. Amitraz should be applied outside or in a well-ventilated area.
2. The recommended dilution of Aludex® (UK) is a 0.05% solution (1:100 dilution equating to 500 ppm). This can be made by adding 50 ml Aludex to 5 litres of warm water.
3. The recommended dilution of Mitaban® (USA) is a 0.025% solution (1:714 dilution equating to 250 ppm). This can be made by adding 10.6 ml Mitaban to 2 gallons of warm water.
4. Stand the dog in a bath and pour the wash onto the skin. Gently work the emulsion into the skin using a soft brush or sponge. Ensure that the whole body area is covered but avoid the eyes and mucous membranes. The dog should be allowed to stand in the excess solution so that the feet are treated. If pododermatitis is present, the feet can be individually bathed in a bowl containing the solution.
5. Remove the dog from the bath and allow it to dry in a warm environment. DO NOT rinse the amitraz off. If possible, the dog can be taken for a short walk to allow it to dry. The dog should not be allowed to lick the solution off the coat.

Dose and route of administration:

1. For the treatment of demodicosis, ivermectin is administered at a dose of 600 µg/kg (0.6 ml per 10 kg) daily. The solution can be given orally in the dog's food.
2. As a precaution against the development of adverse effects, the dose can be gradually increased for the first 6 days. On day one, the dog receives a dose of 100 µg/kg; on day two, 200 µg/kg; on day three, 300 µg/kg; on day four, 400 µg/kg; on day five, 500 µg/kg; and on day six and thereafter, 600 µg/kg.
3. The treatment and monitoring is continued as described under treatment principles above. As with amitraz, 3 to 9 months of therapy may be required.

Major adverse effects:

Ivermectin is extremely toxic to Collies, Collie crosses and herding breeds. It should NEVER be given to these breeds. Ivermectin may also produce sporadic toxic reactions in other breeds. Signs of toxicity include mydriasis, depression, tremor, ataxia, stupor, vomiting, salivation, coma, seizures and death. Most dogs will recover with supportive care (fluid therapy, control of seizures, maintenance of vital functions). Clinicians should obtain written client consent when using products in an extra-label manner.

Milbemycin (not available in UK)

Trade name: Interceptor®.
Formulation: 2.3 mg, 5.75 mg, 11.5 mg and 23 mg tablets.

Dose and route of administration:

1. For the treatment of demodicosis, milbemycin is administered at a dose of 1 mg/kg daily.
2. The treatment and monitoring is continued as described under treatment principles above. As with amitraz, 3 to 9 months of therapy may be required.

Major adverse effects:

Can be used safely in breeds susceptible to ivermectin toxicity. Dogs should be heartworm-negative prior to treatment. Clinicians should obtain written client consent when using products in an extra-label manner.

TREATMENT OF DERMATOPHYTOSIS

Treatment objectives

To eliminate the fungal infection.

Treatment principles

1. Successful treatment of dermatophytosis is best achieved with a combination of systemic and topical therapy.
2. For systemic therapy, griseofulvin is the treatment of choice. Other drugs can be effective but are rarely needed.
3. For topical therapy, a miconazole/chlorhexidene shampoo (Malaseb®) or enilconazole solution (Imaverol®) are the treatments of choice. Other topical therapies can be used but their efficacy is uncertain.

Itraconazole

Trade name: Sporanox®.
Formulation: 100 mg capsules.

Dose and route of administration:

5–10 mg/kg every 24 hours, orally. Absorption is increased if the drug is given with food. Less frequent administration may still be effective.

Major adverse effects:

Not licensed for use in dogs or cats. Itraconazole may cause anorexia, vomiting and lethargy, although not as commonly as with ketoconazole. As with ketoconazole, itraconazole may occasionally cause elevations in liver enzymes. The use of itraconazole in pregnant animals is not recommended.

Terbinafine

Trade name: Lamisil®.
Formulation: 250 mg tablets.

Dose and route of administration:

Cats: 20 mg/kg every 48 hours, orally.
Dogs: 20–30 mg/kg every 24 hours, orally.

Major adverse effects:

Terbinafine may cause gastrointestinal upsets.

Lufenuron

Trade name: Program®.
Formulation: 23.1 mg, 67.8 mg, 204.9 mg, 409.8 mg tablets for dogs; 133 mg and 266 mg ampoules containing suspension for cats.

Dose and route of administration:

Cats: 30 mg/kg once a month, orally, until mycological cure.
Dogs: 10 mg/kg once a month, orally, until mycological cure.

Major adverse effects:

No major adverse effects have been reported. Exact efficacy not known.

Topical products for the treatment of dermatophytosis

The following products can be recommended for topical use in dermatophytosis.

2% Miconazole/2% chlorhexidene

Trade name: Malaseb®.
Formulation: Shampoo.

Dose and route of administration:

1. Instruct the owner or veterinary staff to wear suitable protective clothing (gloves, apron and eye protection).
2. Wet the entire body of the dog or cat with warm water.
3. Apply small quantities of the shampoo to various parts of the body and lather all over. Avoid getting shampoo into the eyes.
4. Massage the shampoo into the skin for 10 minutes. The 10-minute contact time is essential for therapeutic activity.
5. Rinse well and allow to dry in a warm, draught-free environment.
6. Repeat the bath twice a week until mycological cure.

Major adverse effects:

Malaseb® can be irritating to the eyes and contact with the animal's and owner's eyes should be avoided. In case of accidental contact, the eyes should be rinsed with plenty of water. Malaseb® can also occasionally cause skin irritation.

Enilconazole (not available in USA)

Trade name: Imaverol®.
Formulation: 10% concentrated solution (100 mg/ml).

Dose and route of administration:

1. Mix one part of the solution with 50 parts warm water to yield a 0.2% emulsion.
2. Wash dogs with the emulsion four times, at 3–4-day intervals, ensuring each time that the skin is thoroughly wet.

Major adverse effects:

The 0.2% solution of enilconazole is well tolerated. It is not irritating to the eyes or skin and does not cause adverse effects if it is licked by the dog. Although Imaverol has been used successfully in cats, it is not licensed for use in this species.

Lime sulfur (not available in UK)

Trade name: LymDip®.
Formulation: Concentrated liquid for topical use.

Dose and route of administration:

1. Owners and veterinary staff should wear gloves and protective clothing when using this product. Lime sulfur should be applied outside or in a well ventilated area.
2. Add 8 ounces (227 ml) of lime sulfur to one gallon of water.
3. Stand the dog or cat in a bath and pour the wash onto the skin. Gently work the solution into the skin using a soft brush or sponge.
4. Ensure that the whole body area is covered but avoid the eyes and mucous membranes.
5. Remove the dog or cat from the bath and allow it to dry in a warm environment. DO NOT rinse the lime sulfur off and do not allow the animal to lick the solution off the coat.
6. Repeat the application twice a week until mycological cure.
7. Lime sulfur can be used in dogs and cats of any age.

Post-pill testing

Post-pill testing is performed by submitting a serum sample for measurement of total T4 and T3 (see Chapter 13, p. 213 for further details). Post-pill testing is used for two reasons:

1. If the dog has responded well, post-pill testing is performed to ensure that the concentration of thyroxine is not too high. A blood sample should be submitted 4 to 6 hours after the dose of thyroxine has been given. The serum concentration should be at the upper end of the normal range at this time. If the concentration is too high, the dose of thyroxine can be reduced.
2. If the dog has not responded well, post-pill testing is performed to determine if the thyroid hormone concentrations are too low. Two blood samples should be submitted, one taken before the dose is given (trough value) and one taken 4 to 6 hours after the dose of thyroxine has been given (peak value). Both values should be within the normal range. If either value is too low, the dose of thyroxine should be increased or twice daily dosing should be recommended. In some cases, the T3 concentration may be slightly below the normal range whilst the T4 concentration is in the normal range. This does not usually signify therapeutic failure.

What if the treatment fails?

Hypothyroidism may fail to respond to treatment for the following reasons:

Reason for failure	*Solution*
Incorrect diagnosis (the most common reason)	Ensure that the diagnosis is correct (see Chapter 13, p. 212)
Insufficient dose of thyroxine	Check thyroxine concentrations with post-pill testing
Poor owner compliance	Ensure that owner is administering the drug

TREATMENT OF HYPERADRENOCORTICISM

Treatment objectives

To alleviate the dog's clinical signs by medical or surgical therapy

Treatment principles

1. The treatment of canine hyperadrenocorticism is determined by the cause. Adrenal tumours are usually treated by surgical resection. Adrenalectomy requires a highly skilled surgeon backed up by a specialised medical team. The procedure is associated with many intra- and post-operative complications and it is recommended that the case be referred to an institution with expertise in managing this condition.
2. Pituitary-dependent hyperadrenocorticism is usually treated medically with drugs that destroy the adrenal cortex or suppress steroid synthesis. Treatment is required for the remainder of the dog's life.
3. For many years, mitotane (o,p-DDD) was the medical treatment of choice for pituitary dependant hyperadrenocorticism. However, trilostane has recently been licensed for the treatment of hyperadrenocorticism in some countries. In countries where this is the case, this would be the recommended initial treatment, with mitotane being reserved for dogs that fail to adequately respond.

is increased if the drug is given with food. After 14 days, the efficacy should be assessed with an ACTH stimulation test. If satisfactory control of cortisol concentrations has been achieved, the effective daily dose is continued and ACTH stimulation tests are performed every 3–6 months. In occasional cases, doses of 20 mg/kg every 12 hours are necessary.

Major adverse effects:

Not licensed for use in dogs or cats. Ketoconazole may cause inappetence, vomiting, lethargy, pruritus and a reversible lightening of the haircoat. It occasionally causes hepatotoxicity and monitoring of liver enzymes is indicated if the animal appears unwell. Ketoconazole is teratogenic and should not be used in pregnant animals.

L-deprenyl (Seleginine hydrochloride)

Trade name: Anipryl®, Selgian®.
Formulation: 4 mg and 10 mg tablet.

Dose, route of administration and treatment schedule:

L-deprenyl can be used for the treatment of hyperadrenocorticism in cases that are unable to tolerate mitotane, but 50–70% of cases may fail to respond. The initial dose is 1 mg/kg every 24 hours, orally. If there is no response after 2 months, the dose should be doubled to 2 mg/kg every 24 hours. If there is still no response, alternative treatment is necessary. The efficacy of this therapy can only be monitored by clinical signs and ACTH stimulation tests are not useful.

Major adverse effects:

Adverse effects of L-deprenyl are uncommon and include diarrhoea, lethargy, restlessness and disorientation. L-deprenyl is a monoamine oxidase inhibitor and should not be administered alongside other drugs with the same effect (amitraz, tricyclic antidepressants, fluoxetine, alpha-2–agonists). It should not be used during pregnancy or lactation.

What if the treatment fails?

Hyperadrenocorticism may fail to respond to treatment for the following reasons:

Reason for failure	*Solution*
Failure of trilostane therapy	Try alternative medical therapy
Insufficient dose of mitotane	Check cortisol concentrations by ACTH stimulation testing
Dog doesn't tolerate mitotane	Try alternative medical therapy
Failure of ketoconazole or L-deprenyl therapy	Try alternative medical therapy
Incorrect diagnosis	Ensure that the initial diagnosis is correct (see Chapter 13, p. 221)
Poor owner compliance	Ensure that owner is administering the drug

TREATMENT OF MISCELLANEOUS ALOPECIAS

Treatment objectives

To attempt to achieve hair regrowth.

Table 14.11 The prognosis and treatment options for alopecic conditions

Prognosis	Disease	Treatment options
Hair will come back spontaneously	Post-clipping alopecia	None required
	Telogen defluxion	None required
	Anagen defluxion (as long as insult or disease is removed)	None required
Hair may come back spontaneously but alopecia can become permanent. Treatment has been reported but: ● Treatment is for cosmetic, not medical, reasons ● It may be associated with adverse effects ● It may not be effective	Cyclic flank alopecia	Melatonin
	Alopecia areata	Topical or systemic glucocorticoids
Hair will not come back without treatment. However: ● Treatment is for cosmetic, not medical, reasons ● It may be associated with adverse effects ● It may not be effective	Adrenal sex hormone alopecia	Castration Mitotane Testosterone Melatonin
	Testosterone responsive alopecia	Testosterone
	Pattern baldness	Melatonin
Hair may come back with medical treatment. However, the prognosis for full regrowth and a normal coat is poor to fair	Dermatomyositis	Prednisolone Pentoxifylline
	Pituitary dwarfism	Thyroxine Progestogens
	Sebaceous adenitis	Topical therapy Retinoids
	Paraneoplastic alopecia	Surgical removal of pancreatic tumour. Recurrence is likely due to metastasis
Hair will come back with successful treatment. The prognosis for full regrowth and a normal coat is fair to good	Demodicosis	Amitraz
	Dermatophytosis	Griseofulvin Malaseb
	Hypothyroidism	Thyroxine
	Hyperadrenocorticism	Various
	Testicular and ovarian neoplasia	Gonadectomy
Incurable – hair will not grow back	Congenital alopecia	None
	Follicular dysplasia	None
	Colour dilution alopecia	None
	Black hair follicular dysplasia	None
	Epitheliotropic lymphoma	Palliative treatment for other manifestations
	Scars	None

- Keratolytic agents break down the cohesiveness between keratinocytes, improving desquamation and shedding.
- Keratoplastic agents normalise (reduce) the rate of keratinocyte turnover by having a cytostatic effect on basal cells.

Examples of anti-seborrhoeic shampoos are shown in Table 14.12, and described below.

Table 14.12 A guide to choosing anti-seborrhoeic shampoos

Indication	Active ingredients of appropriate shampoo
Normal skin or mild, dry scaling	Emollients (corn, coconut, olive and cottonseed oil; lanolin, paraffin and petrolatum). Humectants (propylene glycol, lactic acid, glycerine, urea and colloidal oatmeal). Essential fatty acids (linoleic and linolenic acid)
Moderate or severe dry scaling	Sulphur and salicylic acid
Greasy scaling	Benzoyl peroxide Tar Selenium sulphide Acetic acid/boric acid
Scaling associated with pyoderma	Benzoyl peroxide Ethyl lactate
Seborrhoea associated with *Malassezia* dermatitis	Miconazole/chlorhexidene Acetic acid/boric acid

When using any anti-seborrhoeic shampoo, the following basic procedure should be adhered to:

1. Instruct the owner or veterinary staff to wear suitable protective clothing (gloves, apron and eye protection).
2. Wet the entire body of the dog with warm water.
3. Apply small quantities of shampoo to various parts of the body and lather all over. Avoid getting shampoo into the eyes.
4. Massage the shampoo into the skin for 10-minutes, especially in the most severely affected areas. The 10-minute contact time is essential for therapeutic activity.
5. Rinse well and allow to dry in a warm, draught-free environment.
6. Repeat the bath two or three times a week during the treatment period, depending on severity.

Emollients/humectants/essential fatty acids

Trade names: Dermocanis®, DermaPet® conditioning, Dermasoothe®, Episoothe®, HydraPearls®, Hylashine®, HyLyt®efa, Humilac®, PearLyt®, Sebocalm®.
Formulations: Shampoos and conditioners.

Dose and route of administration:

Topical application as described above.

Indications and adverse effects:

These shampoos may be used for routine bathing of dogs with normal skin. They are also useful for dogs with mild, dry scaling (seborrhoea sicca) which may be primary or secondary to hormonal imbalances, environmental factors (dry heat) or atopic dermatitis/food allergy (they also remove allergens from the skin). They may also be alternated with, or used after, a more drying shampoo.
No major adverse effects have been reported.

Sulphur/salicylic acid

Trade names: DermaPet® seborrhoeic, Kerect®, Sebahex®, SebaLyt®, Sebamoist® Sebomild®, Sebolux®, SeboRx®.
Formulations: Shampoos.

Dose and route of administration:

Topical application as described above.

Indications and adverse effects:

Used for managing mild cases of seborrhoea sicca not responding to a moisturising shampoo or moderate to severe cases of seborrhoea sicca. Useful for primary idiopathic seborrhoea, sebaceous adenitis, ichthyosis.

Benzoyl peroxide

Trade names: Benzoyl-Plus®, DermaPet Benzoyl Peroxide Plus®, Oxydex®, Paxcutol®, Pyoben®, SulfOxydex®.
Formulations: Shampoos.

Dose and route of administration:

Topical application as described above.

Indications and adverse effects:

Benzoyl peroxide is used for primary idiopathic seborrhoea oleosa, acne, and Schnauzer comedone syndrome. Also indicated for pyodermas (antibacterial action) and demodicosis (follicular flushing improves penetration of amitraz).

Benzoyl peroxide can be irritating to the skin and lead to excessive dryness of the coat. If this occurs, an alternative product should be used. Benzoyl peroxide can also bleach carpets and furnishings.

Tar ± sulphur/salicylic acid

Trade names: Allerseb T®, Clear Tar®, LyTar®, Mycodex Tar and Sulphur®, NuSal T®, Sebolytic®, T Lux®, Tarlite®.
Formulations: Shampoos.

Dose and route of administration:

Topical application as described above.

Indications and adverse effects:

Tar shampoos are used for greasy seborrhoea not responding to benzoyl peroxide or sulphur/salicylic acid. Tar shampoos can be irritating to the skin and can lead to excessive dryness of the coat. If this occurs, an alternative product should be used. Tar shampoos are malodorous, aesthetically unpleasant and can also stain fur or furnishings. Tar shampoos are toxic to cats and should not be used in this species.

Selenium sulphide

Trade names: Seleen®, Selsun Blue®.
Formulation: Shampoo.

Dose and route of administration:

Topical application as described above.

Indications and adverse effects:

Selenium sulphide can be used if other shampoos fail to control a greasy seborrhoea, or if no other products are available. Selenium sulphide is poisonous if taken internally so animals should not be allowed to lick the suspension.

2% Acetic acid/2% boric acid (not available in UK)

Trade name: MalAcetic®.
Formulation: Shampoo.

Dose and route of administration:

Topical application of shampoo as described above.

Indications and adverse effects:

Acetic acid/boric acid shampoos can be used for greasy scaling and seborrhoea associated with *Malassezia* dermatitis. No adverse effects have been reported.

Ethyl lactate

Trade names: Dermacleanse®, Etiderm®.
Formulation: Shampoo.

Dose and route of administration:

Topical application as described above.

Indications and adverse effects:

Ethyl lactate can be used to treat scaling secondary to superficial pyoderma. Ethyl lactate is less irritating and drying than benzoyl peroxide.

2% Miconazole/2% chlorhexidene

Trade name: Malaseb®.
Formulation: Shampoo.

Dose and route of administration:

Topical application as described above.

Major adverse effects:

Malaseb can be used to treat seborrhoea secondary to *Malassezia* dermatitis. Malaseb® can be irritating to the eyes and contact with the animal's and owner's eyes should be avoided.

Dose and route of administration:

1–3 mg/kg every 24 hours

Indications and adverse effects:

Isotretinoin can be effective in the treatment of sebaceous adenitis, Schnauzer comedone syndrome, ichthyosis, feline acne and some skin tumours (epitheliotropic lymphoma, keratoacanthoma). Toxicity in dogs and cats appears to be less of a problem than it is in people. Adverse effects in dogs include keratoconjunctivitis, inflammation of the lips, pruritus, pedal erythema, vomiting, diarrhoea, hyperactivity and joint stiffness. Adverse effects in cats include anorexia, vomiting, diarrhoea and conjunctivitis. Animals should have their tear production checked periodically whist receiving retinoid therapy. Long-term use may induce skeletal abnormalities (corticol hyperostosis, periosteal calcification, osteoporosis). Retinoids are potent teratogens and must not be given to pregnant animals. Female owners of childbearing age should be warned of these potent effects before they handle the drug. Retinoids may also induce biochemical abnormalities including hypertriglyceridaemia, hypercholesterolaemia, and increased liver enzymes, although these are not usually clinically significant. They drugs are very expensive and cannot be routinely prescribed outside of referral centres.

Topical products for the treatment of naso-digital hyperkeratosis

Focal areas of hyperkeratosis on the nasal planum or footpads can be treated with the following products.

Petroleum jelly

Trade name: Vaseline®.
Formulation: Jelly.

Dose and route of administration:

Apply to the affected area twice daily and then as necessary for maintenance.

Major adverse effects:

No major adverse effects have been reported.

6.6% Salicylic acid

Trade name: Kerasolv® (not available in the UK).
Formulation: Gel.

Dose and route of administration:

Apply to the affected area twice daily and then as necessary for maintenance.

Major adverse effects:

No major adverse effects have been reported.

5% Lactic acid/2.5% sodium pyrrolidone carboxylate

Trade name: Lacticare®.
Formulation: Lotion.

Dose and route of administration:

Apply to the affected area twice daily and then as necessary for maintenance.

Retrieval of the foreign body might not be possible at the first visit, especially with grass seeds that can migrate from the site of initial penetration.

4. With tooth root abscesses, the animal needs to be anaesthetised and the tooth removed for healing to occur.
5. With all abscesses, the animal should be treated with a 5–7 day course of an appropriate antibiotic (see below).
6. If the abscess fails to heal, or continuously relapses, it should be treated as a draining tract or non-healing wound and further investigation is required (see Chapter 8 for further details).

Antibiotics for the treatment of abscesses

Ampicillin

Trade name: Amfipen®, Ampicare®, Ampicaps®, Amfipen LA®.
Formulation: 50 mg, 125 mg and 500 mg tablets; 250 mg capsules; 100 mg/ml suspension for injection.

Dose and route of administration:

Tablets and capsules: 10–25 mg/kg every 12 hours, given on an empty stomach.
Injection: 15–20 mg/kg by subcutaneous injection. A second injection can be given after 48 hours.

Major adverse effects:

The drug should not be given to animals, or handled by people, known to be allergic to penicillin or cephalosporins.

Amoxycillin

Trade name: Amoxinsol®, Amoxycare®, Amoxypen®, Betamox®, Bimoxyl®, Clamoxyl®, Duphamox®, Duphamox LA®, Vidamox®.
Formulations: 40 mg, 200 mg, 400 mg tablets; 250 mg capsules; 50 mg/ml oral suspension; 150 mg/ml suspension for injection – depending on manufacturer.

Dose and route of administration:

Tablets, capsules and suspension: 10–20 mg/kg every 12 hours, orally.
Injection: 15 mg/kg by subcutaneous injection. A second injection can be given after 48 hours.

Major adverse effects:

Hypersensitivity to amoxycillin can occur. The drug should not be given to dogs, or handled by people, known to be allergic to penicillins or cephalosporins.

Clavulanic acid potentiated amoxycillin

Trade names: Clavamox®, Synulox®.
Formulation: 50 mg, 62.5 mg, 125 mg, 250 mg, 375 mg, 500 mg tablets; 50 mg/ml oral suspension.

Dose and route of administration:

12–15 mg/kg every 12 hours, orally.

Major adverse effects:

Hypersensitivity to amoxycillin can occur. The drug should not be given to dogs, or handled by people, known to be allergic to penicillins or cephalosporins.

What if the treatment fails?

Abscesses may fail to respond to treatment for the following reasons:

Reason for failure	*Solution*
Failure to drain the abscess	Provide drainage
Failure to remove a nidus of infection (foreign body, tooth root)	Remove nidus
Resistance of the organism to the drug chosen	Change to an alternative drug or perform a culture and sensitivity test
Abscess is caused by an unusual organism (nocardia, actinomyces, mycobacteria, fungus)	Perform tissue cultures (see Chapter 8)

TREATMENT OF DEEP BACTERIAL PYODERMA

Treatment objectives

1. To identify the organisms responsible for the deep pyoderma (usually *Staphylococcus intermedius* but occasionally gram-negative organisms).
2. To successfully eliminate the bacterial infection.
3. To identify and treat the underlying cause to prevent recurrence.

Treatment principles

1. Successful treatment of deep pyoderma is best achieved with a combination of systemic and topical therapy.
2. For systemic therapy, an appropriate antibiotic should be chosen on the basis of culture and sensitivity testing. Examples of antibiotics likely to be useful are shown in Table 14.13.
3. Use an adequate dose based on the dog's body weight.
4. Treat for an appropriate length of time. For deep pyoderma, treatment may last for months, but should be continued for 2–4 weeks past complete clinical cure. Complete clinical cure should be determined by the clinician on the basis of palpation as well as observation.
5. DO NOT USE CONCURRENT CORTICOSTEROIDS, as this will inhibit the body's ability to clear the infection and may lead to recurrence.
6. If possible, treat at the same time with an appropriate shampoo (see later).
7. Treat or control the underlying cause.
8. Reinspect the dog every 3–4 weeks to monitor clinical response. Prescribe continued treatment until completely resolved.

Antibiotics for the treatment of deep pyoderma

Unlike with superficial pyoderma, culture and sensitivity testing should be performed in dogs with deep pyoderma at the first visit. The antibiotics that are likely to be suitable for the treatment of deep pyoderma are listed in Table 14.13 and described below. If a number of antibiotics are shown to be effective on the sensitivity profile, the final choice must be

Table 14.13 Systemic antibiotics for treating canine deep pyoderma

Presentation	Drug
Deep pyoderma caused by *Staphylococcus intermedius*	Cephalexin Cephadroxil Clavulanic acid potentiated amoxycillin Oxacillin
Deep pyoderma caused by *Staphylococcus intermedius* ± gram-negative organisms	Difloxacin Enrofloxacin Marbofloxacin Orbifloxacin

made by the clinician taking into account such factors as cost, ease of administration, required frequency of administration, availability, personal preferences and the following factors:

- Penicillin, ampicillin, amoxycillin and tetracycline are not suitable because the organism is likely to rapidly develop resistant to these antibiotics.
- Clindamycin, lincomycin and tylosin are not good choices for deep staphylococcal pyoderma because resistance may develop during the long course of treatment.
- Potentiated sulphonamides are not good choices because long-term use may induce keratoconjunctivitis sicca.
- In the opinion of many dermatologists, the use of fluoroquinolones for the treatment of deep pyoderma should be reserved for gram-negative, mixed or highly resistant infections.

Cephalexin

Trade names: Ceporex®, Keflex®, Rilexine®.
Formulation: 50 mg, 75 mg, 250 mg, 300 mg, 500 mg, 600 mg tablets, 100 mg/ml oral suspension, depending on the manufacturer.

Dose and route of administration:

22–30 mg/kg every 12 hours, orally.

Major adverse effects:

Hypersensitivity to cephalexin is very rare. The drug should not be given to dogs, or handled by people, known to be allergic to penicillin or cephalosporins. The dose of cephalexin should be reduced in dogs with pre-existing renal disease. Oral administration of cephalexin often causes vomiting in dogs but this usually subsides after the first few doses. The problem is reduced if the drug is given with food.

Cephadroxil

Trade name: Cefa-Tabs®.
Formulation: 50 mg, 100 mg and 200 mg tablets.

Enrofloxacin

Trade name: Baytril®.
Formulation: 15 mg, 50 mg and 150 mg tablets.

Dose and route of administration:

5 mg/kg every 24 hours, orally.

Major adverse effects:

Enrofloxacin should not be administered to rapidly growing dogs as it can cause damage to weight-bearing articular cartilage. This period includes up to 8 months in small dogs, 12 months in medium dogs and 18 months in large dogs.

Marbofloxacin

Trade name: Marbocyl®.
Formulation: 5 mg, 20 mg and 80 mg tablets.

Dose and route of administration:

2 mg/kg every 24 hours, orally.

Major adverse effects:

Marbofloxacin should not be administered to rapidly growing dogs as it can cause damage to weight-bearing articular cartilage. This period includes up to 8 months in small dogs, 12 months in medium dogs and 18 months in large dogs. Marbofloxacin can occasionally cause vomiting, diarrhoea, modification of thirst and changes in activity. Overdosage with marbofloxacin can cause neurological disorders and the drug should be used with caution in epileptic patients.

Orbifloxacin

Trade name: Orbax®.
Formulation: 6.25 mg, 25 mg and 75 mg tablets.

Dose and route of administration:

2.5 mg/kg every 24 hours, orally.

Major adverse effects:

Orbifloxacin should not be administered to rapidly growing dogs as it can cause damage to weight-bearing articular cartilage. This period includes up to 8 months in small dogs, 12 months in medium dogs and 18 months in large dogs.

Topical treatment of deep pyoderma

Topical anti-bacterial therapy is a useful adjunctive treatment in deep pyoderma. However, topical therapy may be impractical in certain circumstances such as:

- With very large or difficult to handle dogs
- With elderly or infirm owners
- During very cold weather when dogs need to be bathed outside.

The following basic principles should be followed when treating deep pyoderma:

Beneficial and adverse effects:

Benzoyl peroxide is a good antibacterial agent that also has a degreasing and follicular flushing action. It has good residual activity. It can be used in all cases of pyoderma but it is the shampoo of choice if the coat is greasy or in cases secondary to demodicosis. However, benzoyl peroxide can be irritating to the skin and lead to excessive dryness of the coat. If this occurs, an alternative product should be used. Benzoyl peroxide can also bleach carpets and furnishings.

Ethyl lactate

Trade names: Dermacleanse®, Etiderm®.
Formulation: Shampoo.

Dose and route of administration:

Topical application as described above.

Beneficial and adverse effects:

Ethyl lactate is a good antibacterial agent with good residual activity. Ethyl lactate is less irritating and drying than benzoyl peroxide.

Mupirocin

Trade names: Bactroban®, Bactoderm®.
Formulation: Ointment.

Dose and route of administration:

Apply to focal areas of deep pyoderma once or twice daily

Beneficial and adverse effects:

Mupirocin is useful for localised areas of deep pyoderma such as acne, calluses, pressure points and interdigital pyoderma. The ointment should not be applied to broken skin. Adverse effects are uncommon.

What if the treatment fails?

A deep pyoderma may fail to respond to antibacterial therapy for the following reasons:

Reason for failure	*Solution*
Choice of inappropriate antibiotic	Use one of the drugs shown in Table 14.13
Underdosing	Calculate the dose based on the dog's bodyweight
Insufficient duration of therapy	Treat for a minimum of 2–4 weeks past clinical cure
Concurrent use of glucocorticoids	Do not administer glucocorticoids when treating pyoderma
Resistance of the organism to the drug chosen	Choose the drug on the basis of culture and sensitivity tests
Unusual organism (nocardia, actinomyces, mycobacterium)	Perform tissue cultures
Incorrect diagnosis	Confirm diagnosis with cytology and bacterial culture. Rule out other causes of draining tracts (see Chapter 8).

What if the pyoderma responds but then relapses?

The most common reason for a deep pyoderma to respond but then relapse a few days to a few weeks after the therapy has been stopped is failure to identify and treat an underlying cause. In such cases, clinicians should rule out:

- Demodicosis
- Atopic dermatitis
- Food allergy
- Hypothyroidism
- Hyperadrenocorticism
- Systemic or neoplastic diseases

The order in which these underlying causes are investigated should be based on the clinician's index of suspicion. If the condition is pruritic after the bacterial infection has resolved, the parasitic and allergic diseases should be ruled out first. If the condition is non-pruritic after the infection has resolved, the most likely causes are demodicosis, hypothyroidism, hyperadrenocorticism, systemic diseases and neoplasia. If all the above conditions have been ruled out and the infection keeps recurring, the clinician can make a diagnosis of idiopathic recurrent pyoderma or breed-specific pyoderma (German shepherd dogs, English bull terriers). Readers should consult the section on staphylococcal pyoderma (page 248) for details on the long-term management of these conditions.

Unusual deep bacterial infections

The bacterial infections described in this section are rare and will not be discussed in detail. The treatment options for these infections are summarised in Table 14.14. If possible, the choice of antibacterial agent should be based on the results of culture and sensitivity testing.

TREATMENT OF DEEP FUNGAL INFECTIONS

Treatment objectives

1. To identify the organisms responsible for the deep fungal infection.
2. To eliminate the fungal infection.

Treatment principles

1. The treatment of deep fungal infections varies depending on the cause.
2. Localised deep fungal infections (mycetoma, phaeohyphomycosis) are best treated by wide surgical excision followed by systemic antifungal therapy.
3. Widespread or systemic deep fungal infections require long courses of systemic antifungal agents.
4. In some cases, drugs need to be used in combination to achieve resolution of the disease.
5. Treatment should continue for 1–3 months after the lesions have completely resolved.
6. DO NOT USE CORTICOSTEROIDS, as this can allow the organism to disseminate with fatal consequences.
7. Some cases of deep fungal infection cannot be cured.
8. Owners and veterinary staff should be warned that some of these infections are zoonotic.

Table 14.15 Treatment options for deep fungal infections

Disease	Treatment options
Eumycotic mycetoma, Pseudomycetoma, Phaeohyphomycosis	Surgical excision, Itraconazole, Fluconazole, Ketoconazole, Amphotericin B
Sporotrichosis	Potassium iodide, Itraconazole, Ketoconazole
Blastomycosis, Histoplasmosis, Cryptococcosis, Coccidiomycosis	Itraconazole, Fluconazole, Ketoconazole, Amphotericin B
Pythiosis	Wide surgical excision

Systemic products for the treatment of deep fungal infections

Various systemic drugs have been used for the treatment of deep fungal infections. The recommended treatment protocols are summarised in Table 14.15 and described below. Currently, itraconazole is the treatment of choice for most deep fungal infections in terms of its efficacy and safety margin. If itraconazole is not effective, a combination of amphotericin B and itraconazole or ketoconazole may be effective. Fluconazole may also be effective either on its own or in combination with amphotericin B. The use of potassium iodide solutions is reserved for the treatment of sporotrichosis.

Itraconazole

Trade name: Sporanox®.
Formulation: 100 mg capsule.

Dose and route of administration:

5–10 mg/kg every 24 hours, orally. Absorption is increased if the drug is given with food.

Major adverse effects:

Not licensed for use in dogs or cats. Itraconazole may cause anorexia, vomiting and lethargy, although not as commonly as with ketoconazole. As with ketoconazole, itraconazole may occasionally cause elevations in liver enzymes. The use of itraconazole in pregnant animals is not recommended. Itraconazole has caused hepatitis and death in one cat.

Fluconazole

Trade name: Diflucan®.
Formulation: 50 mg, 150 mg and 200 mg capsule.

Dose and route of administration:

Dogs: 10–20 mg/kg every 12 hours, orally.
Cats: 50 mg/cat every 24 hours, orally.

Major adverse effects:

Not licensed for use in dogs or cats. Adverse effects are rarely seen.

Ketoconazole

Trade name: Nizoral®.
Formulation: 200 mg tablet.

Dose and route of administration:

10–30 mg/kg every 24 hours, orally, for up to 30 days. Absorption is increased if given with food.

Major adverse effects:

Not licensed for use in dogs or cats. Ketoconazole may cause inappetence, vomiting, lethargy, pruritus and a reversible lightening of the haircoat. It occasionally causes hepatotoxicity and monitoring of liver enzymes is indicated if the animal appears unwell. Adverse effects are more common with doses over 10 mg/kg day. Ketoconazole is teratogenic and should not be used in pregnant animals.

Amphotericin B

Trade name: Fungizone®, Fungilin®, Amphocil®, Abelcet®, AmBisome®.
Formulation: Vial containing 50 mg powder, reconstituted to a 50 mg/ml solution for injection.

Dose and route of administration:

1. Amphotericin B is given as a series of intravenous infusions every 48 hours (usually performed on a Monday–Wednesday–Friday schedule).
2. Prior to the first dose of amphotericin B, a complete blood count, biochemistry profile and urinalysis should be performed. Patients with renal failure, blood dyscrasias or electrolyte imbalances should be treated before amphotericin B is administered. Before each additional treatment, a PCV, total protein, urea, creatinine and urinalysis should be performed. If the serum creatinine has risen to greater than 250 μmol/l or the serum urea nitrogen to greater than 15 nmol/l, the treatment should be discontinued until renal function has normalised.
3. Before each treatment, the patient should be given 50 ml/kg 0.9% NaCl over 3–4 hours to induce a diuresis and minimise renal toxicity.
4. For the first treatment of amphotericin B, a test dose of 0.25 mg/kg should be dissolved in 250–500 ml of 5% dextrose and administered over 2–3 hours. The amphotericin B must not be allowed to contact the 0.9% saline solution as it precipitates in the presence of electrolytes.
5. After the treatment, the patient should be diuresed again with 0.9% NaCl at twice maintenance requirements for 2–3 hours to minimise nephrotoxicity.
6. If the patient tolerates the initial treatment, the procedure should be repeated every 48 hours using a dose of 0.5 mg/kg (dog) or 0.25 mg/kg (cat) until a total cumulative dose of 4–5 mg/kg amphotericin B has been administered.
7. Amphotericin B should be used in conjunction with an azole drug such as ketoconazole or itraconazole to minimise the total dose required.

Major adverse effects:

Not licensed for use in dogs and cats. Amphotericin B is extremely nephrotoxic and can lead to life-threatening acute renal failure. The treatment protocols used should be designed to minimise this complication. The administration of amphotericin B often causes phlebitis at the site of catheter placements, necessitating the use of a number of peripheral veins in order to complete the treatment. In the early stages of treatment, patients may become pyrexic following the treatment. This may be due to the pyrogenic effects of some batches of amphotericin B or it may be secondary to circulating pyrogens released from dying fungal organisms. Other adverse effects include nausea, vomiting, dyspnoea, hypothermia and hypotension although these signs may also be related to the products released by dying fungal organisms.

Potassium iodide

Trade name: Generic.
Formulation: Supersaturated solution.

Dose and route of administration:

Dogs: 40 mg/kg every 12 hours, orally with a fatty food (cream or milk).
Cats: 10–20 mg/kg every 12–24 hours, with food.

Major adverse effects:

Potassium iodide can cause anorexia, vomiting, diarrhoea, and lethargy. In addition, dogs may develop an oculo-nasal discharge, a dry hair coat and skin scaling. Cats may develop hypothermia, twitching and cardiovascular abnormalities.

What if the treatment fails?

Deep fungal infections may fail to respond to antifungal therapy for the following reasons:

Reason for failure	*Solution*
Choice of inappropriate drug	Use one of the drugs shown in Table 14.15
Insufficient duration of therapy	Treat for a minimum of 1–3 months past clinical cure
Failure of single drug therapy	Use an azole in combination with amphotericin B
Resistance of the organism to the drug chosen	Choose an alternative drug
Incorrect diagnosis	Confirm diagnosis with cytology and fungal culture. Rule out other causes of draining tracts (see Chapter 8).
Some fungal infections cannot be cured	Euthanasia

IMMUNOSUPPRESSIVE THERAPY

Treatment objectives

To provide long-term control of the animal's clinical signs with the minimal number of side effects.

Treatment principles

1. The management of autoimmune and immune-mediated skin diseases relies on immunosuppressive therapy. Many diseases that require immunosuppressive therapy

Table 14.17 Prednisolone administration for a 25 kg dog – induction to maintenance therapy

	Day													
	1		2		3		4		5		6		7	
Week	am	pm	am	pm	am	pm	am	pm	am	pm	am	pm	am	pm
1	50	50	50	50	50	50	50	50	50	50	50	50	50	50
2	50	50	50	50	50	50	50	50	50	50	50	50	50	50
3	100		100		100		100		100		100		100	
4	100		**95**		100		**90**		100		**85**		100	
5	**80**		100		**75**		100		**70**		100		**65**	
6	100		**60**		100		**55**		100		**50**		100	
7	**45**		100		**40**		100		**35**		100		**30**	
8	100		**25**		100		**20**		100		**15**		100	
9	**10**		100		**5**		100		**0**		100		**0**	
10	**95**		0		**95**		0		**90**		0		**90**	
11	0		**85**		0		**85**		0		**80**		0	
12	**80**		0		**75**		0		**75**		0		**70**	
13	0		**70**		0		**65**		0		**65**		0	
14	**60**		0		**60**		0		**55**		0		**55**	
15	0		**50**		0		**50**		0		**45**		0	
16	**45**		0		**40**		0		**40**		0		**35**	
17	0		**35**		0		**30**		0		**30**		0	
18	**25**		0		**25**		0		**25**		0		**25**	

Methylprednisolone

Trade names: Medrone V®, Medrol®.
Formulation: 2 mg and 4 mg tablets.

Dose and route of administration:

Induction: 1.5–3 mg/kg/day divided into two doses given 12 hours apart.
Tapering: Gradually, as for prednisolone.
Maintenance: 0.4–1.5 mg/kg every 48 hours.

Major adverse effects:

Methylprednisolone has fewer mineralocorticoid effects than prednisolone and can reduce the severity of polydipsia and polyuria. However, the long-term adverse effects are the same (see above). It is not usually necessary to use this drug in cats.

Dexamethasone/Betamethasone

Trade names: Betsolan®, Norbet®, Opticorten®.
Formulation: 0.25 mg tablets.

Dose and route of administration:

Induction: 0.2–0.4 mg/kg every 24 hours.
Maintenance: 0.05–0.1 mg/kg every 2–3 days.

Major adverse effects:

Dexamethasone and betamethasone are higher potency glucocorticoids than prednisolone and have a longer duration of action. They are less suitable for the treatment of autoimmune and immune-mediated diseases than prednisolone because they are likely to lead to more adverse effects in the long term. Their use should be reserved for cases that have failed to respond to appropriate doses of prednisolone. For adverse effects, see prednisolone.

Azathioprine

Trade name: Imuran®.
Formulation: 25 and 50 mg tablets.

Dose and route of administration:

Induction: 50 mg/m^2 or 2.2 mg/kg every 24 hours, orally, until clinical response.
Maintenance: 50 mg/m^2 or 2.2 mg/kg every 48 hours, orally.

Note: To convert body weight into surface area, see Table 14.18. Because of the available tablet sizes and the undesirability of splitting the drug, it is not possible or necessary to taper the dose of azathioprine in the same way as prednisolone. Once the clinical signs are completely in remission, the daily dose can be given every 48–72 hours.

Major adverse effects:

Azathioprine is extremely toxic to cats and the drug should not be used in this species. The major adverse effects of azathioprine in dogs are bone marrow suppression and gastrointestinal upsets. Diarrhoea occurs in many dogs but this often responds to a dosage reduction or temporary suspension of the drug. Most dogs develop a mild, asymptomatic, non-regenerative anaemia that does not require cessation of therapy. If the total white blood cell count drops to between 5.0–7.0 × 10^9/l, the dose should be reduced by 25%. If the total white blood cell count drops to below 5.0 × 10^9/l, the drug should be discontinued until the

Tetracycline/oxytetracycline: 25 mg/kg every 8 hours, orally OR
Doxycycline: 10 mg/kg every 24 hours, orally AND
Niacinamide: Dogs >10 kg, 500 mg given every 8 hours, orally
Dogs <10 kg, 250 mg given every 8 hours, orally.

Major adverse effects:

The treatment may take 4–6 weeks before an improvement is noticed. The major adverse effects are anorexia, vomiting and diarrhoea, primarily attributable to the niacinamide. Tetracyclines should be avoided in animals with known sensitivities to the drug and in immature animals because they can lead to tooth discoloration. Tetracyclines may also cause oesophagitis and oesophageal ulceration. Tetracyclines can also cause photosensitisation. Tetracyclines should not be used in pregnant animals.

Pentoxifylline

Trade name: Trental®.
Formulation: 400 mg tablet.

Dose and route of administration:

10–15 mg/kg every 8 hours.

Major adverse effects:

Adverse effects are uncommon and include vomiting and diarrhoea.

Dapsone

Trade name: Maloprim®, Alvosulfon®.
Formulation: 100 mg tablet.

Dose and route of administration:

1 mg/kg every 8–48 hours, orally.

Major adverse effects:

Dapsone may cause anaemia, leukopaenia, thrombocytopaenia, hepatotoxicity and cutaneous drug eruptions. These effects are more likely to occur in cats. Dapsone is not licensed for use in dogs or cats.

Danazol

Trade name: Danol®.
Formulation: 100 mg and 200 mg capsules.

Dose and route of administration:

4 mg/kg every 8 hours.

Major adverse effects:

Hepatopathy and cutaneous drug eruptions. Danazol is not licensed for use in dogs or cats.

Vincristine

Trade name: Oncovin®.
Formulation: 1 mg/ml and 2 mg/ml vials for injection.

Dose and route of administration:

1 mg/m^2 (0.01–0.025 mg/kg) given by intravenous injection once weekly.

Ear cleaners and wax dissolvers

These products contain various ceruminolytic and drying agents. They are indicated for:

- Ear canals that are very waxy.
- Ear canals that are too dirty to allow antimicrobial drops to penetrate.
- Long-term management of chronic ceruminous otitis.

Trade names: Epiotic®, Leo Ear Cleaner®, Aurotex®, Auroclens®, Cerumenex®, Cerumene®, Cerusol®, Clear-X®, Surfac®, Oticalm®, Alocetic®, MalAcetic Ear Cleaner®, Oti-Clens®, Otodex (not all available in the UK or USA).
Formulation: Cleaning solutions.

Dose and route of administration:

1. Fill the ear canals with ear cleaning solution.
2. Hold onto the pinna and massage the vertical canal.
3. Leave the ear for 5 minutes.
4. Remove excess ear cleaner with a piece of damp cotton wool or a moistened gauze swab.

Major adverse effects:

Ear cleaners may occasionally cause irritation or allergic reactions in the ear canal. If this occurs, an alternative product should be used. Routine use of ear cleaners in cats should be avoided as they can exacerbate ear canal inflammation. Ear cleaners should be used with care in dogs with a ruptured tympanic membrane as they can lead to ototoxicity.

Antiparasitic ear drops

These products kill ear mites because they contain either antiparasitic agents or ingredients that drown the mites. They are indicated for infestations of *Otodectes cynotis*.

Trade names: Auroto®, GAC®, Otodex®, Oterna®, Canaural®, Aurimite®, Canex®, Cerumite®, Eradimite®, Mita-Clear®, Mitox®, Nolvamite®, Oticare®, Otomite® (not all available in the UK or USA).
Formulation: Ear drops.

Dose and route of administration:

1. Apply four to five drops into the affected ear once daily for 7 days.
2. Suspend treatment for 7 days.
3. Continue the treatment for a further 7 days.

Major adverse effects:

Some antiparasitic eardrops contain glucocorticoids. Topical glucocorticoids are potent anti-inflammatory drugs that can suppress the pituitary adrenal axis and lead to elevated liver enzymes. Prolonged use should be avoided. Owners should avoid contact with the skin. If irritation or adverse reactions occur, the medication should be discontinued.

Selamectin (systemic antiparasitic agent)

This product is indicated for the treatment of *Otodectes cynotis* infestations.

Trade names: Stronghold® (UK), Revolution® (USA).
Formulation: Spot-On for topical use.

Table 14.20 Topical antimicrobial / glucocorticoid ear drops (not all available in the UK and USA)

Trade name	Constituents
Auroto®	Neomycin Thiabendazole Amethocaine
Betsolan eye and ear drops®	Neomycin Betamethasone
Canaural®	Franmycetin Nystatin Prednisolone Diethanolamine fusidate
Canesten®	Clotrimazole
Conofite®	Miconcazole
Forte topical®	Penicillin Neomycin Polymyxin B Hydrocortisone
GAC®	Neomycin Amethocaine Permethrin
Gentocin otic®	Gentamycin Betamethasone
Liquichlor®	Chloramphenicol Tetracaine Prednisolone
Otomax®	Gentamycin Clotrimazole Betamethasone
Oterna®	Neomycin Monosulfiram Betamethasone
Panalog®	Neomycin Thiostrepton Triamcinolone
Pimavecort®	Neomycin Natamycin Hydrocortisone
Surolan®	Polymyxin B Miconazole Prednisolone
Topagen®	Gentamycin Betamethasone
Tresaderm®	Neomycin Thiabendazole Dexamethasone
Tritop®	Neomycin Tetracaine Isoflupredone

5. Tobramycin – tobramycin eye drops (Tobrex®) can be applied directly to the ear canal as a drop.
6. Amikacin – a 50 mg/ml solution of injectable amikacin (Amikin®) can be applied directly to the ear canal as a drop.
7. Acetic acid solution – a 50:50 mixture of white vinegar and water can be effective at killing *Pseudomonas aeruginosa*. This solution is usually used as a flushing or cleaning agent prior to the application of other products. It should not be used in conjunction with aminoglycosides as the acidity can inactivate the antibiotics.
8. Silver sulfadiazine cream – the cream can be diluted with water to make a 0.5% solution and applied directly to the ear canal.
9. TrisEDTA – this solution can be obtained commercially in the USA (TrizEDTA®, Dermapet) or made according to the following recipe:

- 1.2 g EDTA
- 6.05 g Tris buffer
- 1 L of sterile distilled water
- Adjusted to pH 8.0.

This solution potentiates the action of aminoglycosides and possibly fluoroquinolones. It may also be directly bactericidal for *Pseudomonas*. The solution can be used as a flushing or cleaning agent prior to the application of topical antibiotics.

Treatment of otitis media

If the tympanic membrane has ruptured, the clinician is faced with the treatment of otitis media. This occurs most commonly due a long-standing otitis externa in which the infection breaks through the tympanum. However, it can occasionally arise due to a primary infection in the middle ear cavity. If there are no irreversible pathological changes in the horizontal canal or bullae, medical management can be successful. The dog should be anaesthetised and the middle ear cavity flushed out with sterile saline. This is best achieved with a cut-down urinary catheter attached to a 20 ml syringe and a drip set via a three-way tap. The catheter should be extended into the bulla through an otoscope cone or video otoscope. The bulla and ear canal should be thoroughly irrigated, allowing the excess material to flow up and out of the ear. This procedure should be repeated every 2–3 days until the production of exudate has ceased. The infection should also be treated with an appropriate topical and systemic antibiotic based on culture and sensitivity testing. The ear should be checked by cytology every 2–3 days to monitor the levels of microbial infection. Satisfactory treatment is indicated by a reduction in the number of organisms and inflammatory cells, and then their disappearance.

If *Pseudomonas aeruginosa* has been cultured, the treatment is particularly challenging. Currently, the use of fluoroquinolones both topically and systemically at high doses is the treatment of choice. Enrofloxacin should be used at a dose of 15–20 mg/kg and marbofloxacin at a dose of 5.5 mg/kg every 24 hours. This strategy can be effective even if the culture and sensitivity results have indicated resistance. Alternatively, ticarcillin can be used topically and systemically at a dose of 15 mg/kg every 8 hours, given intravenously (the dog needs to be hospitalised). The use of aminoglycosides in dogs with a ruptured ear drum should be avoided as these drugs are known to be ototoxic.

Surgical management of otitis

Surgery can be used as an adjunctive treatment in the management of otitis, or in some cases, it can be curative. The use of surgical techniques in the management of otitis is

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VETERINARY SURGERY

Small Animal Dermatology

Dr Peter B. Hill BVSc PhD DVD DipACVD MRCVS
Director of the Hospital for Small Animals and Senior lecturer in Veterinary Dermatology, Department of Veterinary Clinical Studies, Royal (Dick) School of Veterinary Studies, The University of Edinburgh, UK

- **A unique, practical manual that ensures the correct clinical diagnosis of skin problems in dogs and cats in the most efficient and cost-effective way**
- **Problem-orientated approach takes the reader systematically through the diagnostic process, relating content to clinical veterinary practice**
- **Colour line drawings clearly illustrate the distribution of the diseases**
- **Takes the worry out of performing and interpreting diagnostic tests by explaining which test to use, how to carry it out and how to accurately interpret the results**
- **Separate chapter on treatment provides clear and concise information on managing skin diseases**

Skin disease is one of the most common reasons why dogs and cats are presented to veterinarians and yet dermatological diagnosis and treatment can often prove troublesome. To minimise problems, clinicians must take a logical and systematic approach to carrying out appropriate tests, make an accurate clinical diagnosis and then apply the necessary treatment.

Small Animal Dermatology explains all of the processes involved, taking a problem-based approach to diagnosis and then proceeding to a step-by-step explanation of appropriate tests, highlighting techniques that ensure accurate interpretation of results. Colour line illustrations provide a valuable aid to diagnosis.

Small Animal Dermatology: A practical guide to the diagnosis and management of skin diseases in dogs and cats is an invaluable purchase for practitioners who wish to improve their diagnostic accuracy when dealing with these common conditions, as well as an essential aid for veterinary students who are beginning to learn about skin diseases and their manifestations.

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